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ARCHIVES OF OPHTHALMOLOGY.

ON THE ENDOCRINE ORIGIN OF PRIMARY GLAUCOMA.

By Dr. JOSEPH IMRE, Jr., Budapest,

Professor of Ophthalmology, Royal Elisabeth University, Pécs, Hungary.

(With nine figures in the text.)

OPHTHALMIC research work has few greater problems than that of primary glaucoma. The diagnosis of "glaucoma" must be looked upon as a collective definition of different eye diseases, which have one symptom in common, the increased tension. This causes several other symptoms, which are more or less typical and can be classified in two principal groups.

There is a transition from one group to the other, because we often find that a further increase of intraocular tension in the eye, where the diagnosis of glaucoma simplex is made, can provoke irritation, pain, sudden and further loss of vision, dilatation of the pupil, etc., thus giving the signs of an "inflammatory" glaucoma.

In both groups we have cases, which differ entirely from the general type, yet have to be classified in one or the other group according to their general symptoms, because their principal symptom is the high tension and because up to now we do not know enough to enable us to be more specific.

The permanent and principal symptom, the high tension,

the most fatal consequence of the basal illness, is the cause of most of the other secondary symptoms, but not all. We can prove this by pointing out those changes which are characteristic of glaucoma, but which present themselves independently of the high tension, sometimes long before the increased tension, sometimes after the disappearance of the high tension.

For instance—the glaucomatous pigmentation of the iris, the pigment dust on the posterior surface of the cornea, can be seen long before the other glaucomatous signs (Koeppe).

We know that degenerative processes in the tissue of the iris develop sometimes years after the tension has sunk to the normal.

We must find an explanation that will lift the veil from the secret of those symptoms; but we will err if we try to give a single explanation for all cases. It is one step forward if we accept as a fact that the different forms of glaucoma can have different origins.

The increased tension being the cause of most of the other symptoms, it was quite natural that the ophthalmic investigators have tried to explain the origin of this, hoping thereby to solve the whole problem. The paths they took ran in three directions, sometimes touching each other, sometimes crossing, sometimes diverging. The first is based on pathological physiology, the second on anatomic changes, the third on chemical changes.

Those changes built up on histologic findings are based on changes which are mostly consequences and not causes of the high pressure. The blocking of the angle of the anterior chamber in cases of ***primary*** glaucoma has to be looked upon as a consequence of the high pressure, not to mention those cases in literature where acute glaucoma could be examined histologically and where the angle of the anterior chamber was found free.

As for the sclerotic changes in the arteries, although they may lead in certain exceptional cases to high tension, they are not characteristics of glaucoma and were frequently found also in non-glaucomatous eyes.

Those theories which are based upon hypersecretion caused by irritation of the trigeminus or the sympathetic nerve

(Donders, v. Hippel and Grünhagen, Mooren, etc., Adamück, later Terson, etc.) do not take into consideration the self-regulation of the eye-tension. The same deficiency characterizes the explanation based on high blood pressure. No doubt there were many experiments done which showed that the irritation of the trigeminus or the sympathetic nerve can cause a slight transitory increase of the tension, just as much as the cutting or the paralysis of one of these nerves can cause hypotension, but, as a rule, in both cases the self regulation of the eye tension will be apparent sooner or later.

It was proved half a century ago that any increase or decrease of blood pressure in the big arteries of the body causes corresponding changes in the eye tension. These old statements of Adamück, v. Hippel and Grünhagen, remain valid. The very thorough examinations of Kollner with modern instruments also prove that a close connection exists between the daily rise and fall of the blood pressure and that of the eye tension. These changes of the blood pressure are, however, only *passing* changes, which can only cause similarly passing changes in the eye tension.

Between permanent height of blood pressure and eye tension there is no connection, otherwise patients with the first mentioned trouble would very often get glaucoma and our glaucomatous patients would have very high blood pressure. Leber, himself, thought of this and looked upon the self regulation of the tension as one, the influence of the vasomotor nerves as the other factor, protecting the eyes from glaucoma in cases of very high blood pressure. He thought that the increased tone of the walls of the narrowed retinal blood vessels keep back the effect of the high blood pressure. Wessely explains the normal or decreased tension of elder people by the increased rigidity of the walls of the blood vessels.

These theories are very important, but before further steps are taken it is desirable to know in what degree these statements can explain our clinical experiences.

As I pointed out earlier (1920, 1922) observations have made us doubt the general acceptability of these theories because:—

We know cases where the tension of clinically and functionally normal eyes shows a lasting change without any change

in the blood pressure and without any irritation of the trigeminus or sympathetic nerve.

We were able to show that quite young individuals without any pathological changes in their vascular systems can have ***high*** blood pressure and at the same time very ***low*** eye tension.

We found high intraocular tension in clinically normal eyes, the eyes of individuals who had normal, or, considering their age, relatively low blood pressure.

All these combined show that the regulation of the eye tension and the lasting changes in it cannot be explained simply by changes of the blood pressure and by nervous influences.

We were able to prove, furthermore, that a disturbance in the balance of the endocrine organs causes, as a rule, a ***lasting*** change of the intraocular tension. These changes can be an increase or decrease of the tension, sometimes combined with a great lability of the tension during the measuring. When the balance is restored the intraocular tension becomes quite or almost normal. Therefore we may conclude that the regulation of the intraocular tension depends primarily on the function of the endocrine system. Although the disturbed function of one gland in this system provokes a change in the function of the other glands, and therefore a pluriglandular disturbance is usually present, the glands that seem to play the principal rôle in this regulation are the thyroid, hypophysis, gonads, and adrenals.

We came to this conclusion on finding that hyperfunction of the hypophysis, furthermore, pregnancy with signs of hyperpituitarism go hand in hand with decrease of the eye tension.

Hyperfunction of the thyroid can likewise cause low eye tension. Here, however, we have to emphasize the fact that in Graves' disease either type of abnormal eye tension may be found, but in either case we find, almost invariably, a remarkable difference of tension between the two eyes and a pronounced lability of the tension even during the measuring.

In osteomalacia that is caused by a hyper-dysfunction of the ovaries and perhaps the parathyroid glands, the tension is usually exceedingly low, sometimes lower than that of a cadaver.

In case of pregnancy, where no signs of vicarious hyperfunction of the hypophysis can be found, we find high tension,

sometimes up to 30–32*mm hg.*, without any disturbance of the function of the eyes.

Considering that increased function of the incretory glands is connected with low tension, we may suppose that decreased function of the same glands is combined with high tension. We have a justification of this supposition in the high eye tension of certain pregnant women and of some patients with dystrophia adiposo-genitalis. Patients with a general insufficiency of their endocrine glands show likewise a disturbance of their tension: increase and lability.

This gave me the idea of observing our glaucoma patients from this viewpoint and on the basis of our clinical observations I administered different organic substances on the theory that a good effect can be looked upon as proof of the connection between glaucoma and the deficiency of that particular gland.

The first experiments were very promising although we began our investigations with the necessary scepticism. I began these experiments in 1919 and thus some of our observations date back four years.

Before stating my conclusions a few cases are outlined, to demonstrate the close connection between glaucoma and the endocrines.

The organ extracts used in the following cases were produced by Gedeon Richter, Budapest, who was so kind as to supply the necessary fresh extracts. It is very important that the extracts shall be as fresh as possible, because some extracts, although produced by well-known laboratories, were not quite reliable and I believe that the cause of this lies in the fact that these were not quite fresh products.

It is possible that the method of extraction followed by the Richter laboratory differs from the methods of other factories, therefore I asked the laboratory to tell me their procedure.

The fresh material is extracted on the day when taken from the animal. The organs are cleaned of blood and neighboring tissues; the removal, as well as the whole procedure, is performed in a strictly aseptic way. After this the glands are ground to a pulp. For making tablets the pulp is exsiccated at body temperature and after controlling, their

active contents are compressed into tablets with indifferent vehicles.

For hypodermic injections the pulp is extracted at body temperature with water, the albumen is removed, the quantity of active substances is tested and the material is sterilized again after it is in phials.

The liquids are treated the same way but the albuminous substances are not removed.

Extracts and gland substances used in our experiments:

Tabl. thyreoideæ: one tablet = 0.5 gm. fresh gland.
Extr. thyr. 1*ccm:* 1 gm. fresh gland.
Liquor thyreoideæ, 1*ccm:* 0.25 gm. fresh gland.
Adrenalin tablets (Tonogen Richter) 1 tablet: 0.0005 gm. adrenalin.
Tabl. hypophyseos cerebri 1 tablet :0.3 gm. fresh hypophysis.
Extr. hypophyseos 1 injection: 0.2 gm. gland.
Tabl. ovarii 1 tablet: 1 gm. fresh ovary.
Liquor ovarium, 1*ccm:* 1 gm. fresh gland.
Extract ovariale (Glanduovin) 1*ccm:* 1 gm. fresh gland.
Tabl. testiculi 1 tabl.: 1 gm. gland.
Liquor spermin 1*ccm:* 2 gm. of the fresh testicle of bull.
Extract testiculi 1*ccm:* 1 gm. fresh gland.

Case 1. St. K., age 68, male. He noticed in 1915, that his left eye was weakening. Had no pain, temporary blurring of sight, nor colored rings around the light. His left eye was trephined in 1915, the vision became worse after the operation. His right eye was normal until the spring of 1917, when he noticed a weakening of vision and had difficulty in finding his way about. He was then operated again, anterior sclerotomy was performed on both eyes. From that time on he used pilocarpin but his vision sank gradually.

Status præsens XII. 10, 1919: Right eye: vision with -1.5D. $\frac{6}{60}$, very narrow field. Anterior chamber shallow, iris not atrophic, dark brown with many darker pigment spots. Pupil very contracted.

Left eye: counts finger at 1 meter, very narrow, excentric field of vision. Anterior chamber and iris look like those of the right eye, only there is a basal excision upwards. On the limbus there is a cystoid trephine scar.

Both disks are deeply cupped and white. Retinal vessels are thin, periphery cannot be examined on account of the very narrow pupils. Tension r.: 32, l.: 28 (Schiötz).

Examination of the endocrine system (Dr. Szondi): The thyroid is in a great degree of involution. Isthmus cannot

be palpated, side lobes are very thin, like cords on both sides. Gonads, penis seem normal. Sella turcica slightly enlarged, processus clinoidei are very thick. No shadow of thymus, percussion negative. Lymph system normal. Vegetative and animal nervous systems seem normal. The build of the patient is eunuchoid, face and hands are of acromegaloid type.

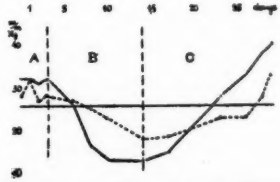

CHART 1. (Case 1.) A. Shows the effect of 2% pilocarpin 3 drops daily. B. Pilocarpin and daily thyroid tablets. C. pilocarpin and 3 thyroid tablets. Right eye —, left eye.....

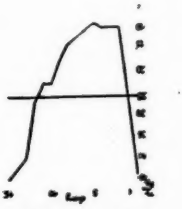

CHART 2. (Case 1.) The effect of cyclodialysis lasts only eleven days.

On the basis of this report we decide to administer first of all thyroid, later other endocrine extracts. The effects of these and of the operation performed during the treatment are shown in diagrams. (See charts 1–5.)

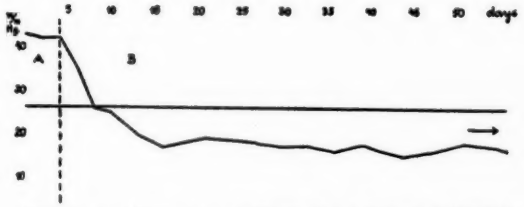

CHART 3. (Case 1.) A. Shows the effect of pilocarpin alone. B. The influence of hypophysis on the tension, 6 tablets daily (= 1.8 gm. fresh gland).

The result is enduring so far as the tension goes. For more than a year there was no higher tension on the right eye than 16*mm.* When I saw him the last time his tension was 12–16, but the vision is sinking gradually. Dilating the pupils by instillation of adrenalin, we found lots of pigment spots in the periphery of the retina. This seems to be very common in cases of glaucoma simplex and a close

connection between the pigmentation of the retina and glaucoma seems to be highly probable.

Epicrisis: The pilocarpin had no influence on the tension worth mentioning. Thyroid could not be administered on account of side effects, although it was effective. The extract of hypophysis had a good and lasting effect. Testicular

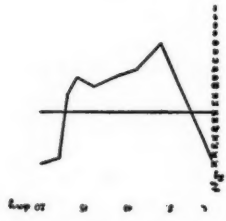

CHART 4. (Case 1.) The effect of a second cyclodialysis lasts but 15 days.

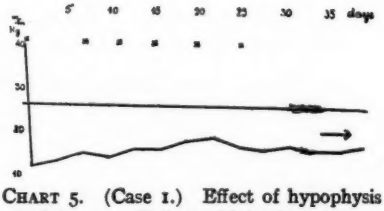

CHART 5. (Case 1.) Effect of hypophysis substance. X days of injections (1 *ccm*)

extract had a strong but not lasting influence on the tension. The organ extracts were at one period of greater value than operation.

The sad circumstance that the vision could not be preserved is explained by the degenerative process of the retina.

CASE 2. T. L., age 23, female, was sent from the school for midwives. Her eyes began to fail when she was 17 years old and she saw colored rings from time to time. She lost the sight of her right eye within 4–5 days in a glaucomatous attack. She was operated a week later, the pains stopped, but no vision came back. Her left eye was operated a few weeks later.

In spite of the operations her eyes were hard and the vision of her left eye also was sinking. Since her pregnancy her eyes feel better and she even stopped using pilocarpin, which she had used for four years.

The right eye has no perception of light. Very deeply cupped chalky white disk; left eye counts fingers at 1.5 meters. Tension in both eyes 18*mm*.

The hospital where she was operated upon kindly gave me the following data concerning her previous condition. She was iridectomized on her right eye on account of acute glaucoma. Two weeks later they performed sclerotomy on her left eye, instead of iridectomy on account of excessive bleeding after the operation of the right eye. A year ago

they proposed another operation because the tension, especially that of the left eye, was constantly increased.

At the time of her coming to us she was in the 7th month of her pregnancy. The signs of vicarious hyperfunction of the pituitary body were apparent, and probably explain the low tension.

CASE 3. E. N., age 50, female. Noticed in 1916, that her eyes are failing. Glaucoma was diagnosed and a basal iridectomy was done on her right eye. In spite of this her vision is often blurred and at the same time she has headaches. V. 27, 1918. Right: vision $\frac{6}{6}$, with convex 0.5 cylinder $\frac{6}{6}$. A narrow coloboma in the iris. Field impaired in the upper nasal quarter. Typically cupped disk. Left: vision $\frac{6}{6}$ E. No conspicuous pathologic signs, the only change which tells that this eye also has glaucomatous tendencies is a quantity of pigment dust on the posterior surface of the cornea and the numerous pigment spots of epithelial origin on the iris. These signs are equally pronounced in the right eye.

Tension: 26–26; pilocarpin was instilled 6 hours before.

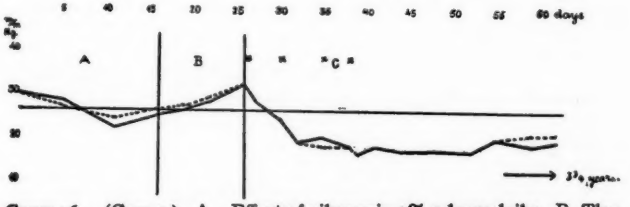

CHART 6. (Case 3.) A. Effect of pilocarpin 2% 3 drops daily. B. Thyroid tablets 3 a day; C. Hypodermics of ovary extract (X) later liquid and tablets.

Next morning tension is 28–29, without pilocarpin. We order pilocarpin 3 times daily. A month later tension is normal.

She returns a year and a half later, complaining about headaches and periodic blurring of her eyes. XII. 18, 1919. Right eye: no outward change, vision $\frac{6}{6}$, with 0.5 cylinder $\frac{6}{7}$. Field is the same as before. Left: $\frac{6}{6}$, field normal. Tension 28–28, although she uses pilocarpin twice daily.

The endocrine examination shows a moderate involution of the thyroid and strong senile involution of the sexual organs.

First thyroid extract was administered, later ovarian extract in form of injection, tablets and drops. The effect of these extracts is demonstrated on Chart 6. The last

examination took place VIII. 30, 1923. No change either in the vision or in the fields. Tension of both eyes *23mm.* No change in the pigmentation of the iris.

Epicrisis: In spite of regular instillation of pilocarpin the tension oversteps the high boundary of the normal. Administering thyroid extract is effective, but cannot be continued on account of side effects. Hypophysis tablets cause *a rise of tension.* Ovarian extract given first in hypodermic form, later in tablets and drops, keeps the tension *within normal limits without pilocarpin.* Period of observation more than 5 years. *No pilocarpin or eserin was given in the last* $3\frac{1}{4}$ *years.*

CASE 4. Sz. I., age 56, female, has been under treatment for retinitis pigmentosa since the age of 14. Has very narrow field since her childhood, but saw very well. She has been gradually losing her vision since her 46th year.

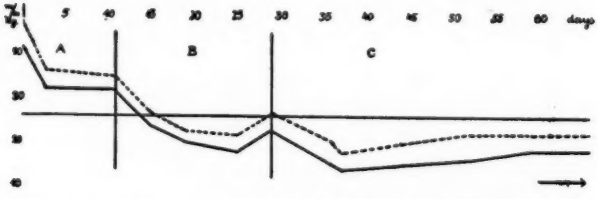

CHART 7. (Case 4.) A. Effect of 2% pilocarpin 5 times daily. B. Decrease of tension; good but not lasting effect of daily 3 tablets ovarium. C. Lasting good result after taking daily 3 thyroid tablets for four weeks, from this on for two weeks in every month.

III. 21, 1920. Right eye: rather small cornea, horizontal diameter 8.5*mm* radius 7.2*mm.* Shallow anterior chamber. In the iris and on the posterior surface of the cornea many pigment spots of epithelial origin (Slit lamp and corneal microscope). Pupil of middle size, reaction sluggish. Retinal vessels are thin, many pigment spots of branching shape along the blood vessels, especially in the periphery. Nearer to the centre there are atypical larger accumulations of pigment. Vision $\frac{8}{60}$, field telescopic and eccentric.

Left eye: under the horizontal diameter of the cornea there are two white spots, the size of a pinhead, superficial and consisting of degenerated epithelial cells. Fundus same as on the right eye. Vision $\frac{1}{60}$, field is restricted to a small temporal area.

Tension: right 42*mm*, left 48*mm*.

Endocrine examination: Very strong involution of the thyroid. Menstruation always irregular lasting usually one or two days, sometimes absent for two months. Adrenalin dilates the pupil to maximum; pronounced sympathicotony.

Chart 7 shows the effect of pilocarpin, alone, given with ovarian extract and with thyroid extract. She takes thyroid tablets every second month for two weeks, daily 3 tablets.

Epicrisis: Degeneratio pigmentosa retinæ followed by glaucoma simplex. Thyroid extract has a strong and lasting influence on the tension.

In this case the arrest of the glaucoma is due to organotherapy. Period of observation 3¼ years.

CASE 5. D. K., 58, male. His vision has been changing daily for the last two weeks. Has headaches and in the evenings he sees colored rings around the lamp. His vision became suddenly very bad a week ago, when he was greatly excited.

IX. 15, 1921. Chambers of both eyes are shallow, pupils vertically oval, somewhat larger than normal, their reaction is hardly noticeable. Vision: right $\frac{5}{50}$, left $\frac{5}{70}$, glasses do not correct. Fields are slightly impaired in the upper nasal quadrant, both sides paracentral scotoma (Bjerrum). Fundus in both eyes hyperæmic, left slight, but typically cupped disk. Tension r. 55*mm*, left 72*mm*.

Five drops of 2% pilocarpin are given daily. Three days later, the pupils are very small, anterior chambers very shallow. He feels better. Vision $\frac{5}{30}$ on the right eye and $\frac{5}{50}$ on the left. Tension 55–70. Operation is advised, but refused.

Endocrine examination finds a conspicuous atrophy of the testicles. Seeing that pilocarpin alone does not bring the tension down to normal, we try first hypophysis extract, later testicular extract, in the beginning combined with pilocarpin drops, later for a short period without them. (See Chart 8). As the patient does not appear regularly and the tension is higher than normal, we try to persuade him to consent to operation but without result. Pilocarpin drops are to be used twice daily and testicular tablets every second month.

Last examination XI. 17, 1923. Has full vision in both eyes and the tension is r. 22*mm*, l. 23*mm*.

This was a typical case of inflammatory glaucoma. Pilocarpin was administered originally only as a preparation for operation. As the patient did not consent, we turned to the organotherapy, partly as an interesting experi-

ment, partly because we expected some good effect. The testicular extract was decided effective. Pilocarpin alone did not work satisfactorily, only after testicular extract had been given for a few days. The extract alone was almost as effective as when given with pilocarpin. He now uses ¼% pilocarpin twice daily and this is enough to keep his tension down. Period of observation: 22 months.

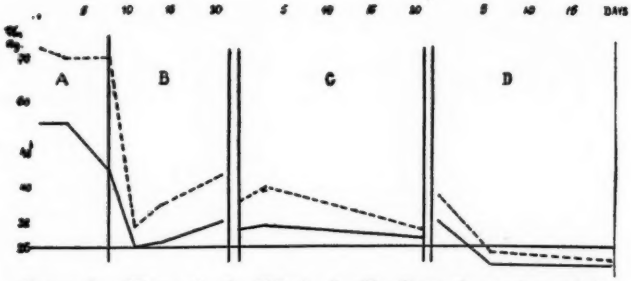

CHART 8. (Case 5.) A. Effect of 2% pilocarpin, 5 drops daily. B. Effect of pilocarpin + daily 6 tablet of hypophysis substance. C. 1% pilocarpin 3 drops daily + 6 tablets of testiculus substance. D. ½% pilocarpin and 30 drops of testiculus substance.

CASE 6. M. St., age 27, female. In the spring of 1920, just at the time of her menstrual period she had a glaucomatous attack. Her physician ordered pilocarpin and her eye recovered in 4 days, and she stopped the drops. Since this attack she has had pain and blurred vision in one or both eyes every month one or two days before her menstruation. A well-known eye specialist advised operation, which was refused.

Her husband, a gynecologist, states that her menstruation is very weak and irregularly, with cramps and hemicranias. She was pregnant three years ago, during which time she had no hemicrania. She had to stop nursing soon and not many months after labor, the first glaucomatous attacks began.

The examination does not show anything abnormal except a few pigment spots on the cornea (slit lamp and microscope). Vision ¾, tension *22mm.* Field free, no Bjerrum scotoma. Fundus slightly hyperæmic.

Ovarian extract is prescribed in liquid form and she is asked to come at her period. Three weeks later, menstruation began and the tension in both eyes was *15mm without pilocarpin.* She was seen again three months later

after she had had four menstrual periods without any glaucomatous signs although no pilocarpin was used, but she had taken ovarian extract.

The next month, June 15, she complained of pain and blurring. V. r.: $\frac{6}{9}$, l.: $\frac{6}{6}$. Tension 20–29*mm*. She had not taken the ovarian drops and did not use pilocarpin. I give her hypodermic injections of ovarian extract. Twenty-four hours later the tension was 16*mm* in both eyes. Vision $\frac{6}{6}$. The next month passed without another attack.

August 23d she has pain in spite of pilocarpin, five drops a day. She did not take any ovarian extract that month.

I saw the patient seven months later in April, 1923. Up to the last month she has taken ovarian extract every

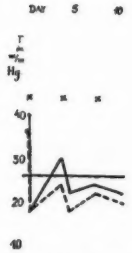

CHART 9. (Case 8.) The effect of hypodermic injections of testiculus extract.

month two weeks previous to the time of her menstruation and her eyes are all right. She is pregnant, now in the second month, and is worried about the future of her eyes. Both eyes are perfectly sound, tension in both 15*mm*.

In June there was a typical acute inflammatory glaucoma attack in her right eye. Pregnancy was interrupted in May. She had an attack one day before coming to me, her vision rapidly grew worse from hour to hour in spite of pilocarpin. Vision r. $\frac{3}{60}$, left $\frac{6}{6}$, field could not be examined on account of her weak condition. Tension, r. *60*, l. *26*. Injection of ovarian extract was given. Next day vision, r. $\frac{6}{24}$, tension *40–20mm*.

Second injection next day; tension, r. *32*, l. *18mm*. Vision *r.*$\frac{6}{9}$. After the third injection the tension fell to 24 on the right and remains 18 on the left. Vision $\frac{6}{6}$ in both eyes (Chart 9.)

EPICRISIS. This case demonstrates quite convincingly that the disturbance of an endocrine gland can cause in-

flammatory glaucoma and also demonstrates the remarkable effect of hormoni therapy.

It was an interesting coincidence that I was consulted in a similar case just about the time the former case was again under my observation.

CASE 7. Female, 22 years old, was iridectomized on her right eye for glaucoma simplex. In the operated eye there is a tumescent cataract which developed within a few weeks after the operation. The left eye was in perfect order but her oculist has noticed periodical increase of the tension. Conservative therapy is advised because the pilocarpin had kept the tension of the left eye within normal limits.

A few months later the same doctor called me again to see his patient. He had performed iridectomy a week previous and there was a plastic iritis. This disappeared soon after two milk injections but later her eyes were irritatable and hard at her periods in spite of pilocarpin, therefore I suggested ovarian tablets which she has taken from the summer of 1922 on. Since then there has been no trouble during her periods. Her left eye has full vision and normal tension.

CASE 8. B. K., age 26, physician. Had cholera in the war. Since then he was unable to regain his weight, is very tall and thin, feels healthy but becomes tired easily. After his illness he has had asthenopic complaints. After ten or fifteen minutes' reading his eyes blur, he sees colored rings around the flame and he has paroxysmal headaches. An eye specialist ordered pilocarpin but the patient used it only a few times because it caused pain and cramps in both eyes, lasting several hours. He even saw colored rings after the pilocarpin.

August 2, 1920, both eyes are slightly reddened. Episcleral veins are visible. Right eye—dark brown iris, pupil larger than medium, reaction somewhat slow, retinal vessels hyperæmic, disc typically but not deeply cupped and quite red. Vision $\frac{8}{8}$—with convex 0.5D. cyl. 20,$\frac{8}{8}$. Field normal, no paracentral scotoma. Tension 36*mm.*

Left eye externally looks the same as right, retinal vessels are strongly filled, disc very red with sharp margins, there is no glaucomatous cup. Vision and refraction are the same as in the right eye, field free. Tension 24*mm.*

Internal examination—except sympathicotony and slightly underdeveloped testicles, no conspicuous abnormality.

Two days after hypodermic injection of thyroid extract the tension is higher 38–33*mm*, although the patient has no disagreeable feelings. Two weeks later, he had headache and blurred vision since last evening caused by sexual excitement. He says this is the rule during the last two years. Tension, both eyes, 40*mm*. He reports certain peculiar observations which are of interest.

During the attack he becomes myopic, even when he has blurred vision he sees quite clearly at a distance of 20*cm*. If he reads for a longer time he notices the same symptoms. During an attack the pain is *worse if he tries to read.* Pilocarpin causes very painful cramps. He feels as though his eyeballs were hardened by the pilocarpin. As these phenomena are in discordance with our knowledge I made a few experiments on him.

His intraocular tension was, at the beginning of the examination, 40–32. After reading very small printing for ten minutes, tension 40–36. Vision with *concave* 0.5D. cyl. $\frac{1}{2}$. otherwise with convex 0.5D. cyl. Next day—Tension 36–30. Massage of the eyeballs for five minutes resulted in an increase of the tension to 40–33. Refraction—changes after massage from convex 0.5D. cyl. to concave 0.25 combined with 0.5 cyl.

This can only be explained by a high degree of hyperæmia and swelling of the ciliary body, which causes the temporary change in the refraction. The increase of the tension is due to the great quantity of blood in the uveal and retinal vessels, as the whole fundus shows very full arteries and veins.

This also explains why the many attacks did not cause any lasting impairment of the central or peripheric vision. The cause of loss of vision is chiefly the retinal ischæmia. The greater intravascular pressure resists the increased intraocular pressure and thus the retina suffers much less than in the case of contracted vessels.

The injection of testicular extract results in a falling of the tension in five hours from 40–36 to 18–18 (See Chart 10), An injection was given every four or five days for two months and he feels quite well. During this time even sexual excitement passes without noticeable change in his eyes.

Two months later he gets an attack again and this time the testicular injections have only a slight effect, the tension sinks from 48–36*mm* to 44–30 in one day. We wish to see how adrenalin acts and first give 3 drops of adrenalin in both eyes. In 30 minutes almost maximal mydriasis. Tension falls to 38–32. We order adrenalin tablets 6 a day. Next morning he reports that the pains disappeared and in the evening he could read without any fatigue. Tension 28–22. He takes the adrenalin tablets for 6 days, at the end of this

period colored rings, headaches and blurred vision. Tension 40–32.

We return to the testicular extracts. Two injections result in reducing of the tension to 28–26 in two days. From this time on he used testicular extract in liquid and in tablets for several months and has no further serious trouble. I am sorry to say, that just before I left Europe, he came back to our clinic with a very severe attack, and this time neither the adrenalin nor the testicular extract had any effect. As the miotios proved to be ineffective also, I urged operation, which was performed a few days before this manuscript was finished and I cannot report on the end result. Four days after the iridectomy the eye was hard and sensitive.

This case shows the connection between the endocrines and juvenile glaucoma, and the decidedly good, but not lasting effect of the testicular extract and the good, but not lasting effect of adrenalin on his tension. The extract of the hypophysis which he took for several weeks worked almost as well as the testicular extract but the effect lasted only for a few weeks. Thyroid extract was positively disadvantageous.

These and other cases, thirty-one in all, justify our belief that in a very large percentage of primary glaucomas constitutional anomalies can be considered as basal causes of the symptoms. In an overwhelming majority, twenty-seven out of thirty-one, the close connection of endocrine disturbances and glaucoma is made highly probable.

Examination gave, in several cases, strong evidence of an abnormal state of the endocrine system. It showed not only the physiologic regressive metamorphosis of this system which is explained by age but in most cases one or more glands proved to be especially degenerated or underdeveloped.

The effectiveness of organotherapy, although it is sometimes only transitory, can be looked upon too as a proof of this relation. The supposition of Esch that it could be considered as a non-specific protein therapy, can be easily refuted because we found the organ extracts effective not only in the form of hypodermics (the extracts we used for injections do not contain albumen) but also when administered as tablets or in liquids. Esch says that "the same extracts are recommended in diametrically opposite conditions." "He treated a series of cases by intra-muscular injection of either breast-milk or certain

proprietary articles (aolan and caseosan) of albuminous nature. His results were certainly as good as, or better than, those to be obtained with the use of organ extracts." (Novak, *Endocrineology*, vol. vi.)

We accept the fact that the same organ extracts can have a good effect on either hyperfunctionating or hypofunctionating glands. The hyperfunction of an endocrine gland is not identical with *increased normal function*. On the contrary it is more probable that the hyperfunction is, as a rule, abnormal function which results in more incretion but in an imperfect one. Some components of the very complicated incretes may be missing and this imperfect production may lead, directly or indirectly, to increased incretion. Clinical observations give us hints which make this explanation probable. For instance, those instances of Graves' disease where signs of hyper- and hypofunction of the thyroid can be found in the same patient.

If hyperfunction does not mean merely a perfect but increased function, if we accept the possibility of incomplete incretion, we cannot deny the possibility of a *regulating* effect of organotherapy. An argument for the validity of this deduction is Case 1 in which, in spite of the signs of hyperpituitarism, the extract of the pituitary body promptly gave good results.

We saw Cases 3 and 4 where the increase of the tension began when the ovaries stopped working, others (Cases 6 and 7) where the acute attacks of glaucoma regularly appeared at the time of imperfect menstruation. In both groups organotherapy was successful. The substitution or regulation of the ovarian function goes hand in hand with the regulation of the tension.

Other cases (1 and 4) demonstrate the connection of involution of the thyroid gland with increase of the tension. The therapy proved our contention and the ocular tension sank to normal.

Of course there are cases where the pathologic changes in the anatomic structure of the eye are already so advanced that the endocrine therapy is unable to check the process.

There are cases where neither the endocrine examination nor the clinical signs gave any proof of a disturbance of the

endocrine system. In two of these there was a slight transitory improvement after administering thyroid extract, but in most of them there was no effect at all. These negative results seem to be just as convincingly in favor of our assertion, that *the effect of the organ secretion is a specific one.*

The last mentioned cases belonged to the group where acute or chronic glaucoma is combined with serious degenerative changes of the entire vascular system, or with serious disturbances of the circulation, caused by spastic conditions of the arteries, which are well known to all ophthalmologists.

These, however, are not usual and there are proofs enough in literature that the average blood pressure of glaucomatous patients is not higher than that of other persons of the same age and changes in the vascular system are only slightly more frequent. This slight increase in numbers is given by those atypical and mostly malignant cases just mentioned.

Statistics which illustrate the relative high percentage of glaucoma in hypermetropic individuals are not convincing. In younger individuals, up to 35 years, 32.4 percent. of glaucoma simplex was observed in myopic eyes and 26 per cent. of the inflammatory glaucoma was combined with myopia (C. Haag).

Axenfeld pointed out recently the possibility of a greater frequency of glaucoma simplex in myopic eyes. He laid emphasis on the great lability of the tension of several myopic patients and the abnormally high tension without conspicuous signs of glaucoma.

The theory of Priestley-Smith, based upon the very small circumlental spaces, cannot be looked upon as of general validity because Hess demonstrated in two cases of primary glaucoma that the circumlental space was larger than normal and there was not the least sign of "swollen," "erigated" or "hyperæmic" ciliary processes.

I have found several times glaucoma in eyes with high degree of hypermetropia. Two patients from the same family were under my treatment, both had H. above 10D with microcornea and malignant glaucoma simplex. A whole family is at present under my observation, the mother having 14D. of H. and in the time of the climacterium she got glaucoma simplex. Two of her sons and one daughter have H. around 10D. All

of them have microcornea (diameter between 7.5 and 8.5*mm*. Radius of cornea between 6.9 and 7.2*mm*).

These eyes could be looked upon, in the sense of Priestley-Smith's theory, as predisposed to glaucoma on account of their probably small circumlental spaces. I, too, consider them predisposed to glaucoma, but on account of their constitutional anomalies, which led to an imperfect growth of the eyeball, and which in a later period of their lives may lead to a lasting increase of their intraocular tension.

As we have every right to say that normal development and growth are ruled by the incretory system we may rightly suppose that abnormal growth of certain organs is caused by a disturbance of that system.

Symptoms of an endocrine disturbance are very common in glaucomatous persons. There is, for instance, the increased tonus of the sympathetic nerve which was found very often in our glaucoma patients.

> Adrenalin causes mydriasis in case of diabetes, Graves' disease, or as A. Loewy showed, is generally a sign of hyperglykæmia. Hyperglykæmia itself is a consequence of endocrine disturbance. Four to five drops of adrenalin given every three or four minutes caused a decided mydriasis in ninety per cent. of our glaucoma patients. Arnold Knapp was the first who published convincing data concerning adrenalin mydriasis in glaucomatous persons, a series of sixty-five cases of primary glaucoma. He found a regular or eccentric dilatation of the pupil in sixty of the sixty-five cases, varying from one to five *mm*. He also measured the tension and found that it was unaffected in forty, decreased in twenty and increased in five. He found, furthermore, "that in fifteen of these 65 cases subjected to adrenalin the glaucoma was only apparent in one eye. The other (normal) eye showed in 12 a dilatation of the pupil after adrenalin, in three there was no change. . . . This suggests that a susceptibility to adrenalin may be present long before the usual clinical signs of glaucoma."

My experience accords perfectly with these findings. The positive reaction in the other normal eye seems to argue in favor of the probability that the effect of adrenalin is not based on a local change only.

We found a positive Loewy reaction in eyes which had no

other signs of glaucoma except the many fine pigment spots described by Koeppe. ***The wandering of pigment and positive adrenalin reaction together can be looked upon as sure signs of a preglaucomatous condition.***

It was only natural that a possible connection between glaucoma and the endocrine system had been thought of by several writers. With the method of Abderhalden, v. Hippel, in 1915 found in glaucomatous patients signs of dysfunction of the thyroid and thymus. These data make systematic examination of glaucoma cases with the improved method of Abderhalden desirable. In a few cases it gave us prompt answers (Büchler). Hertel found hypofunction of the thyroid on his glaucoma patients. Paltracca thinks that glaucoma is a symptom of a disturbance of the organism caused by climacterium. Poyales supposes that predisposition to glaucoma as well as the provoking motives are based on irritation of the sympathetic nerve by adrenalin. Terrien, in accordance with Hertel's and my data, found the disturbance of tension in disturbances of the thyroid. He accepts Hertel's supposition, that they are caused by the change of salt quantity of the blood. Fuchs, in his paper read before the Medical Association in Baltimore, admits that the hyperproduction of adrenalin causes high blood pressure and, through this, high intraocular tension.

Concerning the influence of organ extracts on eye tension, Hertel found that thyraden has a conspicuously good effect on glaucomatous eyes. He came to the idea of administering thyroid by observing a dog that had signs of Graves' disease and had a very low eye tension. He examined patients with Graves' disease and found their tension, as a rule, lower than normal. On the other hand, he found in his patients with glaucoma simplex hyperthyroidism. He explains the increase of tension by changed osmotic conditions.

My examinations concerning the influence of the different endocrine glands on the tension proved that hypotension in Graves' disease is not a rule. I found just as often increased tension as hypotension. I found another writer who suspected an endocrine gland as a regulating organ of the eye tension. This was Cyon who, twenty-five years ago, supposed that this was the hypophysis.

Our observations, however, proved that we have to take into consideration the entire endocrine system. Sometimes one, sometimes another gland can be primarily disturbed and can lead to a change of the intraocular tension but this effect is not necessarily a direct one.

But what may be the cause of the change of tension in these disturbances?

The very thorough experiments of Hertel have shown that a change in the concentration of the blood influences the tension. He showed that intravenous injection of hypertonic salt solution results in a decrease of the eye tension. This effect is more pronounced on animals fed with thyroid substance and is less conspicuous in animals deprived of their thyroid. He supposes that the thyroid has an influence on the osmotic processes by an animating effect on the activity of cells. The increased function of the thyroid gland would therefore explain a quicker exchange between the eye fluids and the blood. A deficiency in this gland could arrest this exchange. Possibly a reduced activity of the cells and of the osmotic processes explains why the intraocular fluid in glaucoma patients contains more albumen as Troncose has found.

There is another observation that makes Hertel's supposition plausible. Ascher found that the red corpuscles in the blood of glaucoma patients show a much quicker sedimentation than those in normal blood, but if he mixed salt solution to glaucomatous blood the sedimentation required more time. This acceleration of the sedimentation is found in various disturbances of the endocrine system.

A change of the osmotic processes could be caused—as Hertel admits—not only by the decreased salt percentage of blood but also by other factors. The big molecules of albumen can not be observed easily and this leads gradually to a qualitative change of the aqueous. All these may happen, without the necessity of endothelial damage as Hertel believes because, if they were present in every case, lasting results would not follow miotics, operative treatment or treatment with organ extracts.

SUMMARY. Individuals with glaucoma have in an overwhelming majority of cases a disturbance of the endocrine system.

Organotherapy often has a decided specific effect on the eye tension of these persons.

In cases in which the endocrine system was found normal organotherapeutic experiments had negative results.

Organotherapy is the more effective the earlier the case is treated, but in many cases there is no lasting effect.

We do not expect that organotherapy will prove miotics and operations superfluous but in several cases one or both may be avoided, and in certain other cases we can increase their effectiveness by administering organic extracts.

Investigations of endocrinologists must be followed, and clinical observations concerning the constitutional changes in our glaucoma patients must be collected, we may then hope to answer the great question—which persons are predisposed to glaucoma and what must be done to prevent this disastrous disease.

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CYCLECTOMY, A NEW OPERATION FOR GLAUCOMA.[1]

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ABOUT two years ago the idea occurred to me that removal of a piece of the ciliary body might reduce the intraocular pressure in cases of glaucoma. This idea was based on the following considerations: (1) The well known fact that injuries and inflammation of the ciliary body have a marked tendency to lower the intraocular pressure. (2) Drainage into the subchoroidal space might be established by the operation. (3) Drainage might take place through the scleral wound if the uveal tissue was removed from its vicinity. (4) The vascular conditions in the ciliary body might be altered in such a way as to reduce the blood pressure therein. (5) Section of some of the ciliary nerves might reduce the intraocular pressure. (6) The operation might reduce the intraocular pressure in some unknown way, just as iridectomy lowers the pressure in glaucoma in some way not certainly known.

I have now performed the operation nine times on seven eyes. The results have been so successful that I am now reporting them in the hope that other operators may also employ the operation and thus determine its exact value more quickly than will be possible for me alone with the relatively small number of suitable cases at my disposal. The operation is performed as follows: The eye is first anæsthetized, either by instillation or by subconjunctival injection of cocaine. Adrenalin is instilled to control hemorrhage. A conjunctival flap, including all of the tissue down to the sclera, is made on the

[1] Read before the New England Ophthalmological Society, Dec. 18, 1923.

upper outer quadrant of the globe, with its base towards the equator. When held back it should expose the sclera from a point about 1*mm* from the corneal limbus for a distance of about 7*mm*. A suture is passed through the conjunctiva and the flap, ready for tying. With a sharp large knife needle—a Post knife needle is especially suitable—an incision about 5*mm* long and parallel to the ora serrata is made through about half the thickness of the sclera at a distance of 5*mm* from the corneal limbus. To avoid injury to the pars optica retinæ, which possibly might cause separation of the retina, it is important not to make the incision further back. The anterior edge of the scleral wound is now grasped with fixation forceps and the scleral incision carefully deepened until the black uvea is seen. This is the most delicate part of the operation and great care must be exercised not to cut through the ciliary body. The blade of the knife is now reversed and the remaining scleral fibers cut from within out by pushing the point of the knife beneath them. The posterior part of the ciliary body will now balloon out through the wound. With a small keratome or the knife needle, a small incision is now made exactly at the limbus above and a small peripheral iridectomy ("buttonhole") made in the iris. If an iridectomy has previously been done, the anterior chamber is simply evacuated by paracentesis. Owing to the reduction in intraocular pressure the prolapsed ciliary body will now recede to some extent. The knife needle or a spatula is introduced, on the flat, under the anterior lip of the scleral wound and the ciliary body separated from the sclera for a short distance. (It is probable that this step is unnecessary.) The protruding part of the ciliary body is now grasped with fixation forceps, with the bite parallel to the wound, pulled out slightly, and excised with scissors. The remaining part will immediately retract into the wound. If vitreous presents, some of it is excised. The wound is covered with the conjunctival flap, the suture tied and additional sutures inserted as needed to insure firm closure. The additional sutures are important as the subsequent œdema may otherwise open the conjunctival wound. Both eyes are bandaged for twenty-four hours, after which no bandage is required. For about a week after the operation, free drainage from the eye takes

place which causes the conjunctiva over the wound to bulge out, sometimes to a marked degree. This need occasion no alarm, as the bulging always subsides.

In all cases the operation was carried out essentially as just described except that in my first case the peripheral iridectomy was not done until three days after the cyclectomy, and in my second case no vitreous was removed. In my first case, the day after the cyclectomy the anterior chamber was alarmingly deep, evidently due to the iris and lens acting as a valve and thus allowing the pressure in the anterior chamber to become higher than in the vitreous chamber.[1] To remedy this condition, three days after the cyclectomy I performed peripheral iridectomy, after which the anterior chamber resumed its usual depth. (It is possible that such temporary deepening of the anterior chamber may be advantageous, especially in cases in which the chamber was originally very shallow. Conceivably it might in this way be possible to free the root of the iris from the pectinate ligament. If necessary for the purpose a forcible injection might be made into the anterior chamber by means of a hypodermic syringe.[2])

In my second case the evacuation of the anterior chamber enabled me, by exercising great care, to perform the cyclectomy without loss of vitreous. In all the other cases I have intentionally removed a small amount of vitreous humor, so as to rupture the hyaloid membrane and thus possibly provide freer drainage of fluid through the vitreous.

It will be observed that the part of the ciliary body removed in this operation is the posterior part, that is the part in which the blood vessels are all small, and which does not bear any of the ciliary processes. While close to the vitreous it is really separated from the latter by zonular fibers. The small size of the blood vessels accounts for the fact that in no case did any observable hemorrhage take place from the cut edges of the ciliary body. The pieces of ciliary body removed at six operations were examined microscopically. In each case the tissue was found to consist of the pars plana only and did

[1] I have seen this condition follow scleral puncture for separated retina.

[2] I have employed this latter procedure in cases of blocked pupil and obliterated anterior chamber, preliminary to iridotomy. A scleral puncture was of course also necessary.

not include any of the ciliary processes or the pars optica retinæ.

In two cases (Cases 4 and 6) it was necessary to repeat the operation at a different site before the tension remained sufficiently reduced. If necessary the operation could be done in each of the four quadrants of the globe. Further experience may show that in certain cases it may be better to make the scleral incision longer or to excise or punch out a piece of sclera in addition.

If it should be found that the reduction in pressure after this operation is mainly due to seepage of fluid through the scleral wound, it may prove advisable in cases in which further reduction in pressure is necessary, later to trephine the sclera over the site of the original cyclectomy, rather than to do a second cyclectomy.

In Case 1 the tension was reduced from 50*mm* (Souter) to 11*mm* (Souter), the reduction persisting throughout the whole period of observation, eleven months. This seems a remarkable and convincing result.

In Cases 2 and 3 the tension was reduced from over 40*mm* (Souter) to 20*mm* (Souter). The latter is somewhat higher than I regard as desirable. No doubt a second cyclectomy would have reduced the tension below 16*mm*. In Case 5, glaucoma following sympathetic uveitis, no other operation seemed to me to offer any hope of success, and the successful result obtained by cyclectomy is therefore especially noteworthy. In Case 6, it is improbable that a complete iridectomy would have lowered the pressure sufficiently, and moreover, the resulting coloboma would have been a serious cosmetic blemish in a young woman. A trephine operation might have been successful, but it would have left the eye liable to late infection.

In only one case, Case 7, was the result of the operation unsatisfactory. This was in a case of hemorrhagic glaucoma in which the patient refused enucleation and insisted on an attempt being made to save the eye. An expulsive choroidal hemorrhage occurred in this case, but the microscopic examination of the eye, which was removed three days later, showed that this did not take place at the site of the operation, but from the opposite side of the eye. In other words, this result

would have followed any operation which involved opening the eye. Serial cross sections of the optic nerve confirmed the diagnosis of hemorrhagic glaucoma by showing complete obstruction of the central retinal vein.

Case 6, in which glaucoma of high degree resulted from contusion of the globe, brings up the question of the cause of the increased intraocular pressure in such cases. Owing to the extensive diffuse hemorrhage into the vitreous in this case the possibility suggests itself that this may have caused the increased pressure. In my opinion, however, this possibility is excluded by the facts that the anterior chamber was abnormally deep, and at no time contained a large amount of blood. To cause glaucoma the blood in the vitreous must have either impeded filtration through the vitreous, or obstructed the spaces of Fontana. In the former event the anterior chamber would have been shallow, in the latter, blood in considerable amount must have been present in the chamber. Moreover glaucoma not infrequently results from contusion of the globe in the absence of intraocular hemorrhage. After contusion of the globe, even without rupture of the latter, diminution in intraocular pressure also often occurs, in fact, probably more often than elevation of pressure. An explanation, to be satisfactory, therefore, must apply to both of these conditions. Two possible explanations suggest themselves. One of them is that the contusion directly injures some of the intraocular vessels and thus alters the intraocular circulation so that in some cases there is a localized increase in capillary pressure, in others a decrease. That the vascular system is affected by the contusion is obvious from the fact that sometimes, as in Case 6, there is intraocular hemorrhage. The other possible explanation is that some of the intraocular nerves are injured, so that in some cases, vasoconstriction, in others vasodilation results. That the intraocular nerves are often injured by a severe contusion of the globe is shown by the fact that more or less complete mydriasis may result. It may be of moment in this connection, that permanent reduction of tension in Case 6 was obtained only after cyclectomy had been done near the site of the contusion.

Owing to the general belief that injuries to the ciliary body are especially likely to cause sympathetic uveitis, many

operators may fear to do this operation. The part of the ciliary body directly affected by the operation, however, contains little if any more uveal tissue per unit area than the iris, and the part remaining in the eye after cyclectomy is less severely traumatized than is the iris by an iridectomy. It is my belief, therefore, that there is less danger of sympathetic uveitis after this operation than after iridectomy.

The chief danger possibly connected with the operation, it seems to me, is postoperative separation of the retina. If this is likely to occur, however, it would seem that it should have occurred in Case 6, in which the operation was performed twice and vitreous removed each time. While the amount of vitreous removed was actually small, it was relatively large compared with that removed in the other cases. As shown by Case 2, loss of vitreous can be avoided, at least in some cases. To avoid it, however, makes the operation more difficult, and I shall continue to make no effort to do so until I find that loss of vitreous in this operation is harmful.

If the results obtained by cyclectomy prove to be permanent, it will be the operation of choice for all cases of chronic glaucoma, since it does not disfigure the eye, interfere with the pupil, or produce a cystoid condition at the limbus which renders the eye liable to late infection. In the meantime I shall continue to employ it only in cases similar to those reported.

Report of Cases

Case 1. Lea D., aged 56.

Chronic glaucoma O.U. with some pain and redness O.S. Has had frequent attacks of "neuralgia" over O.S. for which she has been treated by her family physician.

V.O.S. $\frac{1}{10}$. Pressure O.D. 38*mm*. O.S. 50*mm* (Souter)

Feb. 9, 1923	O.D. Iridectomy by Dr. Riemer.
Feb. 13, 1923	O.S. Cyclectomy by Dr. Verhoeff. No iridectomy. Slight loss of vitreous.
Feb. 14, 1923	O.S. Marked elevation of conjunctiva over wound. Anterior chamber very deep.
Feb. 16, 1923	O.S. Anterior chamber alarmingly deep. Dislocation of lens feared to be imminent. Operation: Small peripheral iridectomy above.

Feb. 18, 1923	O.S. Anterior chamber slightly shallow.
Feb. 24, 1923	O.S. Anterior chamber slightly shallow. Tension normal. Slight œdema of conjunctiva over site of scleral wound. Discharged from Hospital.
May 7, 1923	O.S. Eye white and painless. Pressure normal. Slight œdema over site of scleral wound.
Jan. 3, 1924	There has been no neuralgia since operation. Cyclectomy wound O.S. visible as translucent line. Anterior chamber O.S. shallow, about same depth as that of O.D. Tension O.D. 11*mm*. O.S. 11*mm* (Souter).

CASE 2. Andrew C., aged 59.

Chronic glaucoma O.D. with slight congestion. Deep cupping of disk. Anterior chamber shallow. Tension 42*mm*. (Souter). Visual field greatly contracted.

Vision $\frac{10}{10}$ with glass. O.S. Normal, Vision $\frac{10}{10}$ tension 14*mm*.

May 8, 1923	O.D. Cyclectomy and small peripheral iridectomy. No loss of vitreous.
May 15, 1923	Marked œdema of conjunctiva over scleral wound. Tension 16*mm* (Souter). Discharged from Hospital.
June 7, 1923	V.O.D. $\frac{10}{10}$ +
June 21, 1923	Eye white. Slight œdema of conjunctiva over scleral wound. Tension 20*mm* (Souter).

CASE 3. John O., aged 59.

Chronic glaucoma O.U. Tension O.D. 46*mm* O.S. 32*mm* (Souter).

Vision O.D. fingers at 2½ feet, O.S. $\frac{10}{10}$

June 9, 1923	Operation: O.D., Cyclectomy with small peripheral iridectomy. O.S., Iridotasis.
June 13, 1923	O.D. Marked œdema over scleral wound.
June 16, 1923	O.D. Œdema much less. Discharged from Hospital.
June 22, 1923	O.D. Tension 17*mm* (Souter).
Oct. 12, 1923	Tension O.D. 20*mm*, O.S. 12 *mm*. V. O.D. fingers at 5 feet. The scleral wound appears as a translucent line and the conjunctiva over it seems to show very slight diffuse œdema.

CASE 4. Lester V., aged 21.

Nine years ago was told he had a cataract in right eye. No history of injury. Has had slight pain in O.D. at times. Three days ago had severe pain and eye became inflamed.

Examination: Cornea hazy. Extending from limbus below there is a small gray elevation—slight bulbous keratitis. The iris shows a small hemorrhage on its surface. Lens milky white—hypermature cataract. Vision O.D. shadows, projection good. Tension 20*mm*. O.S. Normal; Vision $\frac{20}{15}$. Tension 10*mm* (Souter).

May 11, 1923	Linear cataract extraction with peripheral iridectomy. Milky lens matter completely removed.
May 25, 1923	Discission of capsule.
May 26, 1923	Discharged from Hospital.
May 31, 1923	O.D. Cornea steamy. Tension 42*mm*. Pilocarpin Nitrate 2% t.i.d.
June 4, 1923	O.D. Tension 22*mm* (Souter).
June 11, 1923	Tension 30*mm*. V. O. D. $\bar{c}$ + 7.50$_s$ = $\frac{20}{100}$.
June 12, 1923	Operation: Cyclectomy up and out; with slight loss of vitreous.
June 25, 1923	Tension 18*mm* (Souter). V. O.D. $\bar{c}$ + 8.25$_s$ = $\frac{20}{25}$ Diffuse œdema of conjunctiva over scleral wound.
July 16, 1923	Tension 38*mm* 2% sol. Pilocarpin Nitrate.
July 23, 1923	Tension 30*mm* (Souter). Operation: Cyclectomy up and in.
July 28, 1923	Tension low normal. Vision O.D. $\frac{20}{30}$ with glass. Discharged.
Dec. 10, 1923	Patient writes that his eye is soft ordinarily, but thinks that its tension is elevated on damp days. As the patient while under observation has always been able to detect increase in tension this statement is probably correct.

CASE 5. Albert C., aged 10.

June 6, 1921	O.D. Perforating wound of cornea and lens with prolapse of iris.
June 7, 1921	Excision of prolapsed iris.
July 1, 1921	Enucleation O.D.
July 7, 1921	Slight congestion O.S.

July 9, 1921	Definite sympathetic uveitis O.S.
July 11, 1921	Treatment with Coley's toxins begun.
July 18, 1921	Treatment with diphtheria antitoxin begun.
Oct. 19, 1921	O.S. white and quiet. Iris adherent to lens everywhere. Delicate pupillary membrane. Cornea clear. Vision $\frac{20}{50}$ $\bar{c}$ glass.
Nov. 7, 1921	Tension 14*mm* (Schiotz).
Dec. 29, 1921	Cornea clear. Tension 28*mm* (Schiotz) V. $\bar{c} - 6_3 = \frac{20}{40}$.
Nov. 4, 1922	Tension 36*mm*. Eye congested.
May 15, 1923	Has had recurring attacks of increased tension, and cornea now shows well marked band keratitis. Anterior chamber normal depth. Operation: Attempted iridectomy. Iris could not be removed from lens, but communication with posterior chamber above lens apparently established.
May 21, 1923	Tension minus.
June 16, 1923	Tension 30*mm* (Souter) not reduced by pilocarpin. V.O.S. $\frac{20}{50} - \bar{c}$ glass. Operation: Cyclectomy up and out.
June 18, 1923	Bead of vitreous presenting from under conjunctival flap, excised.
June 27, 1923	V. O.S. $= \frac{20}{200}$ Discharged from Hospital.
July 2, 1923	V. O.S. $= \frac{20}{200}$.
July 16, 1923	Tension 12*mm* (Souter).
July 30, 1923	V. O.S. $\frac{20}{50}$ Tension normal.
Aug. 6, 1923	V. O.S. $\bar{c}$ glass $\frac{20}{40} - 1$ Tension 25*mm* (Schiotz).
Sept. 6, 1923	Unchanged. Going to school.
Oct. 15, 1923	Unchanged.

CASE 6. Adeline T., aged 27.

May 30, 1923, while riding horseback was struck in right eye by limb of tree. Dr. —— found a tear in the conjunctiva from limbus to inner canthus and some tendon and scleral tissue torn away. The wound was sutured and atropine prescribed. There were only "a few small intraocular hemorrhages." June 3 "a hemorrhage appeared in the anterior chamber and the entire vitreous became very cloudy." June 5 "the hyphema was almost absorbed but there was no fundus reflex." June 16, the intraocular pressure became very marked. June 21, the tension was 42*mm* (Schiotz). "Blood again appears in the anterior

chamber just below edge of pupil, not in lower part of chamber."

June 23, 1923	The patient came under my care. The right eye was considerably congested. Cornea hazy. Anterior chamber free from blood, definitely deeper than that of left eye. Pupil dilated *ad maximum.* Lens clear. Vitreous cloudy. Fundus invisible. Tension 40*mm* (Souter). Vision, light perception with good projection. Left eye normal, Vision with glass $\frac{10}{10}$. Sol. 2% pilocarpine every 2 hours O.D.
June 25, 1923	Pupil contracted to half normal size. Tension 38*mm* (Souter). Paracentesis performed.
June 27, 1923	Pupil smaller. Cornea clear.
June 29, 1923	Tension + Peripheral iridectomy performed.
July 5, 1923	Cornea hazy. Tension 35*mm* (Souter).
July 6, 1923	Cyclectomy up and out. Small amount of turbid dark greenish vitreous removed. (Microscopic examination of this later showed vitreous infiltrated with disintegrating blood —no inflammatory cells.)
July 16, 1923	Conjunctiva elevated over wound. Cornea clear. Tension 27*mm* (Souter).
July 18, 1923	Cornea hazy. Tension 31*mm* (Souter).
July 19, 1923	Subconjunctival sclerotomy below.
July 25, 1923	Tension 15*mm.*
July 30, 1923	Tension 30*mm* (Schiotz).
July 31, 1923	Cornea hazy. Tension 34*mm* (Souter). Cyclectomy in upper inner quadrant. Some turbid vitreous removed.
Aug. 2, 1923	Cornea clear. Tension minus.
Aug. 9, 1923	There seems to be some vitreous beneath conjunctiva. Tension minus.
Aug. 13, 1923	Tension 10*mm* (Souter). Counts fingers at certain place in outer field. A red reflex can be seen through vitreous, corresponding to this place.
Sept. 4, 1923	Tension 11*mm* (Souter).
Sept. 29, 1923	No miotics have been used since last cyclectomy. Tension 11*mm* (Souter). Cornea clear. Pupil only slightly dilated. Both scleral incisions visible, conjunctiva slightly œdematous (?)

over them. Counts fingers at 2 feet, straight ahead. The right eye is now divergent about 20△.

Dec. 22, 1923 Tension 11*mm* (Souter). No congestion. Pupil slightly larger than that of O.S. Fundus now visible, normal. No cupping of disk. Vitreous shows large streaks of old blood anteriorly. Wound of first cyclectomy is plainly visible as a translucent line. Conjunctiva over this may be slightly œdematous. Second cyclectomy wound not now visible.

V. O.D. $\frac{20}{100}$ c̄ + 50_s = − 1.50 cyl. ax. 135° = $\frac{20}{40}$ − 1

Visual field. O.D. normal.

Laterophoria. Right hyperphoria 2△.

CASE 7. Charles C., aged 51.

O.D. Hemorrhagic glaucoma. Vision Nil.

O.S. Normal.

Enucleation O.D. refused.

July 20, 1923 Cyclectomy O.D. Operation difficult owing to hemorrhage from conjunctival and scleral vessels. Intraocular hemorrhage after excision of prolapsed ciliary body.

July 23, 1923 Enucleation O.D.

Microscopic examination of the eye showed complete obstruction of the central vein, and a destructive subchoroidal hemorrhage which evidently took place from the side opposite the operative wound.

THE TONIC EFFECT OF THE SYMPATHETIC ON THE OCULAR BLOOD VESSELS

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(*With one figure in the text.*)

IN a previous paper (1) one of us confirmed the work of Henderson and Starling (2) on the effects of stimulating the cervical sympathetic. We obtained more direct evidence than had formerly been given that the sympathetic contains vaso-constrictor fibers to the blood vessels of the eye, and that stimulation of the head end of this nerve causes a narrowing of the intraocular blood vessels.

It is well known that the intraocular pressure, which ranges normally from 15 to 25*mm* Hg, varies with changes in blood pressure, and this coupled with the facts above stated suggested two questions to us.

1. Is there a tonic influence of the sympathetic on the ocular blood vessels, whereby these vessels are normally kept in a semi-constricted condition?

2. Is this tonic influence effective in preventing changes in the blood pressure from exerting their full effects on the eye where they might cause deleterious changes in intraocular pressure?

Many investigations have been carried out in answer to the first question. The results have been contradictory. They range from no constant effect at all (3), to the rather startling findings of Angelucci (4) and Obarrio (5) who claim that after extirpation of the superior cervical ganglion in young animals,

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not only is there a lowering of intraocular pressure, but even marked trophic changes, such as retarded development of the whole eye, alopecia of the eyelids, etc. We can find in the literature no experimental evidence which has been brought to bear on the second question, although some authors speak most freely of the normal control of the size of the blood vessels which the sympathetic nerves effect. Magitot (6) describes in detail certain nerve cells found among the capillaries of the chorioid which be believes are of the nature of a peripheral sympathetic ganglion connecting the capillaries with sympathetic nerve control. This ganglion is supposed to be keeping the intraocular blood vessels under constant control, and to be effective in preventing changes in the general blood pressure from affecting intraocular pressure, by keeping the blood vessels of the eye at a constant caliber. No experiments are cited, however, which support this hypothesis.

That the sympathetic when stimulated artificially can be effective in preventing changes in general blood pressure from reaching the eye has been proved by Wessely (7), who found that by faradization of the cervical sympathetic or by injection of adrenalin subconjunctivally he could keep the intraocular pressure from rising as high in the experimented eye as it did in the opposite eye when the general blood pressure was raised. These experiments are no proof, however, that under normal conditions the sympathetic left to itself is effective in this manner when the general blood pressure is raised, and we wished to determine this point because of its practical significance.

METHODS.

Cats were used in all experiments, anæsthetized with ether and then given urethane by stomach tube. A loose ligature was placed around the aorta above the superior mesenteric artery, so that by constricting the aorta the general blood pressure might be raised. The intraocular pressure was measured by an instrument essentially like that described by Wessely (7). The pressure was registered by a small Marey capsule covered with the finest rubber membrane obtainable and measuring *7mm* in diameter. A fine glass writing point was used to record the pressures on the smoked drum. The mem-

branes were carefully tested that no leak existed. No records were used where there had not been a completely satisfactory introduction of the needle in the anterior chamber; *i.e.*, where neither the iris nor the lens had been touched in the insertion and where we were positive there was no leak around the needle. We found it helpful regarding this last point to introduce the needle obliquely through the cornea so that the needle passed through approximately 1 to *2mm* of cornea before entering the anterior chamber. In this way we obtained a water-tight junction. Time was allowed after the introduction of the needle for the eye to recover from the immediate effects of the trauma.

The blood pressure was measured by a mercury manometer in either the opposite carotid artery or one of the subclavian arteries and was recorded on the drum by a float riding on the mercury.

EXPERIMENTS CLASS A.

After making sure that the intraocular and blood pressures were recording satisfactorily the blood pressure was raised suddenly, allowed to reach an even level, and then dropped suddenly. This was repeated five times in succession at three minute intervals between each rise to allow readjustment of the eye to the changes in pressure. We usually found, however, that at the end of one minute both blood pressure and intraocular pressure had come into equilibrium. In some cases the vagi were cut on both sides, care being taken not to disturb the cervical sympathetic which lies in close proximity to the vagus in the cat, but separated from it. This was done to get rid of the depressor effect when the blood pressure was raised. After the fifth rise and fall of blood pressure the sympathetic on the side experimented on was cut and five more rises and falls of blood pressure recorded in the manner just described. The intraocular pressure was then calibrated on the drum, the record varnished and measurements made of the corresponding heights of blood pressure and intraocular pressure at synchronous points. No records were accepted where there was an apparent lag in the change of intraocular pressure, which might have been caused by some resistance in the system, as for instance, a small clot. In all cases the

intraocular pressure reached its maximum synchronously with the blood pressure. Ten such experiments were carried out in this manner.

Six controls were made raising and lowering the blood pressure ten times as in the previous experiments but without cutting the cervical sympathetic. This was done as an added precaution to reveal any possible effect on the blood vessels produced by the constant stretching incident to the changes in pressure, although we found as the tables will show that the changes occurred immediately after section of the nerve. The following record is an example of these experiments.

EXPERIMENT A 1.

October 25, 1923—Cat, anæsthetized with ether and urethane. Blood pressure left carotid artery. Intraocular pressure right eye. Left vagus and sympathetic cut. Loose ligature around aorta. Right vagus out. Three minute intervals between rises.

RISES

Blood Pressure Rose		*Intraocular Pressure Rose*	
From	To	From	To
124	166	23.25	30.50
96	146	19.25	26
108	156	19	26
96	154	17.50	24.50
100	154	17.25	24
Average 104.8	155.2	Average 19.23	26.2

Blood Pressure Rose 50.4*mm* Hg

Intraocular Pressure Rose 6.97*mm* Hg

CUT RIGHT CERVICAL SYMPATHETIC

Blood Pressure Rose		*Intraocular Pressure Rose*	
From	To	From	To
124	186	23	33
102	180	21	32
104	186	19.25	32.25
108	192	20	33.50
100	190	19.25	34.75
Average 107.6	186.8	Average 20.5	33.1

Blood Pressure Rose 79.2*mm* Hg

Intraocular Pressure Rose 12.6*mm* Hg

Before the cervical sympathetic was cut the intraocular pressure rose 6.97*mm* Hg for a blood pressure rise of 50.4*mm* Hg. We then calculated what this value should be on the basis of the blood pressure rise after section of the sympathetic by the simple proportion given below. For proof that this calculation is justified see page 245. Additional proof is obtained from the fact that in some experiments the rise in blood pressure after section was lower than before section, and does not influence our results. We find the value should be 10.9*mm* Hg.

Experimental Value = 12.6*mm* Hg
Calculated Value = 10.9
Difference = 1.7

Formula:

$$\frac{\text{Rise in Intraocular Pressure before Section}}{\text{Rise in blood pressure before section}} = \frac{X}{\text{Rise in blood pressure after section}}$$

Where X = Calculated Value of the rise in intraocular pressure after section

Thus it will be seen from these experiments that after section of the sympathetic the intraocular pressure rose on an average 1.7*mm* more than it did prior to section for the same rise in blood pressure. In other words the sympathetic prior to section had been effective in preventing an increase of 1.7*mm* Hg pressure in the eye.

There are two other possible factors besides vaso-dilatation which must be considered when the sympathetic is cut:

1. Constriction of the pupil. When the sympathetic is cut a myosis is always produced on that side. According to the work of Seidel (8) and others, it has been definitely proved that constriction of the pupil allows of freer escape of aqueous from the anterior chamber. If this is effective in our experiments it would tend to lower pressure rather than raise it and so would detract from rather than aid in the results we obtain. No fall of pressure was observed, however, following section of the nerve in our experiments.

2. The effect of Tenon's capsule. If one stimulates the sympathetic the orbital muscle in Tenon's capsule contracts and so raises the intraocular pressure. If this muscle has any tonic effect, which has never been proved, section of the nerve would give a slightly lower pressure in the eye. We have

never observed this and if it did occur it would also, like the myosis, prevent, rather than aid, us in obtaining our results.

In this record as in all subsequent ones we fail to find any effect of cutting the sympathetic on the height of the intraocular pressure at normal blood pressures. We find no evidence that at normal blood pressure the sympathetic is effective in altering the pressure in the eye. It is possible that after a long period of time the myosis produced by cutting the nerve may give a lowered intraocular pressure. This has been reported clinically in numerous cases, but is not, however, a constant finding (9). In all clinical cases the pressure readings are taken with a tonometer, and it is possible that the enophthalmos produced by lesions of the sympathetic may be the factor giving lower tonometric readings,—the atrophy of the

TABLE 1

RISES

Exp. No.	Before Section		After Section		*Rise Calculated mm Hg*	*Difference mm Hg*
	Average B. P. Rise mm Hg	*Average I. P. Rise mm Hg*	*Average B. P. Rise mm Hg*	*Average I. P. Rise mm Hg*		
A 1	50.4	6.9	79.2	12.6	10.9	1.7
A 2	53.5	3.0	55.8	4.8	3.7	1.1
A 3	69.4	4.6	69.3	6.6	4.6	2.0
A 4	84.6	13.7	87.6	15.3	14.2	1.1
A 5	65.6	4.6	81.8	7.3	5.7	1.6
A 6	91.0	7.2	91.6	8.5	7.3	1.2
A 7	62.6	5.2	50.4	5.5	4.1	1.4
A 8	67.6	4.0	69.6	5.8	4.1	1.7
A 9	86.4	7.2	89.4	9.0	7.4	1.6
A 10	54.0	5.5	64.0	7.0	6.5	0.5

Rise in Intraocular Pressures as compared with artificially produced blood pressure changes before and after section of the cervical sympathetic.

orbital muscle making the external coats of the eye less resistant to any pressure applied from the outside.

Table 1 gives a summary of ten experiments including A–1. For the sake of simplicity we have given only the average blood pressure and intraocular pressure rises before and after section of the sympathetic. We then give the rise as calculated on the formula presented above and in the last column show the difference which represents the effectiveness of the sympathetic in each experiment.

The difference in the height to which the intraocular pressure rose before and after section ranged from 0.5*mm* Hg in experiment A–10 to 2.0*mm* Hg in experiment A–3. Table 2

TABLE 2

CONTROL EXPERIMENT

	RISES 1ST GROUP		RISES 2ND GROUP			
Exp. No.	*B. P. Rise mm* Hg	*I. P. Rise mm* Hg	*B. P. Rise mm* Hg	*I. P. Rise mm* Hg	*Calculated Rise mm* Hg	*Difference mm* Hg
A 1	60.0	5.2	52.7	5.1	4.6	.5
A 2	66.8	5.9	62.6	5.2	5.5	− .3
A 3	68.4	5.2	67.6	4.0	5.1	−1.1
A 4	77.6	7.5	86.4	7.2	8.3	−1.1
A 5	43.4	3.9	54.0	5.5	5.0	.5
A 6	54.4	7.7	50.7	7.0	7.1	.1

Rise in intraocular pressures as compared with artificially produced blood pressure changes. Group 1 and 2 are carried out under identically the same conditions as a control.

shows the same values as given in Table 1 for the control experiments. The difference in the control experiments ranges from − 1.1*mm* Hg to + 0.5. *mm* Hg.

Thus when we compare two series of five rises each the normal variation will be within the range of − 1.1 to + 0.5.

When, however, we compare similar series before and after section of the nerve the range lies between + 0.5 to + 2.0.

	Experiment No.	*Difference in intraocular pressure in 1st and 2nd series of rises*
Control experiments	A 3	−1.1 *
	A 4	−1.1
	A 6	+0.1
	A 2	+0.3
	A 1	+0.5
	A 5	+0.5
Experiments in which cervical sympathetic was cut	A 10	+0.5
	A 2	+1.1
	A 4	+1.1
	A 6	+1.2
	A 7	+1.4
	A 9	+1.6
	A 8	+1.7
	A 1	+1.7
	A 3	+2.0

Table 3 shows the same figures for the falls in pressure from the high levels after cutting the sympathetic. The values range from 0.7*mm* Hg to 4.2*mm* Hg. Comparing this with Table 4 which gives the figures for the control falls in blood pressure the values here range from − 2.82*mm* Hg to + 0.6*mm* Hg.

These values simply confirm what we find for the rises. Since, after section, the intraocular pressure rose higher due to increased dilatation of the vessels, when the blood leaves these vessels the total fall of intraocular pressure will be greater.

In some of the experiments it was noticed that the level from which the blood pressure rose each time was different. Because of the prolongation of the anæsthetic the blood pres-

* These minus values are probably evidence that the continued stretching of the blood vessels makes them constrict more and more, and so the intraocular pressure is lower in the 2nd series than in the 1st when the sympathetic is intact.

TABLE 3

FALLS

Exp. No.	Before Section		After Section		Rise Calculated	Difference
	Average B. P. Falls	*Average I. P. Falls*	*Average B. P. Falls*	*Average I. P. Falls*		
A 1	70.2	8.5	93.2	15.5	11.3	4.2
A 2	76.0	5.75	77.0	7.6	5.8	1.8
A 3	82.6	6.05	81.3	6.65	5.95	0.7
A 4	98.6	15.95	90.9	15.46	14.70	.76
A 5	85.6	6.2	93.2	8.65	6.77	1.88
A 6	101.0	8.6	103.6	10.1	8.7	1.4
A 7	65.2	4.9	70.1	6.35	5.25	1.1
A 8	71.2	4.15	57.6	6.15	3.35	2.8
A 9	99.4	10.0	102.8	13.7	10.4	3.3
A 10	78.4	7.1	85.6	8.5	7.7	.8

Fall in intraocular pressures as compared with fall in blood pressures from an artificially raised level, before and after section of cervical sympathetic.

sure usually fell gradually and with it the intraocular pressure by an equal amount. At first we thought that possibly this might be influencing the experiments and that our results were obtained because of this factor. Later we were able to note the same results in animals whose blood pressure remained constant from the beginning to the end of the experiment. It seemed advisable to know what the relationship between intraocular pressure and various heights of blood pressure might be. Wessely (7) pointed out that there was always a disproportion between the percentage rise in blood pressure and intraocular pressure. He states that this disproportion is smaller at the lower pressures and greater at the higher pressures. By inducing a hyperæmia in one eye by a

TABLE 4

CONTROL EXPERIMENT

FALLS 1ST GROUP			FALLS 2ND GROUP			
Exp. No.	*B. P. Fall*	*I. P. Fall*	*B. P. Fall*	*I. P. Fall*	*Calculated Fall*	*Difference*
A 1	62.4	5.3	63.0	5.94	5.33	.61
A 2	77.6	7.2	65.2	4.9	6.0	−1.1
A 3	72.0	7.05	71.2	4.15	6.97	−2.82
A 4	93.4	10.6	99.4	10.0	11.2	−1.2
A 5	75.0	6.6	78.4	7.1	6.9	.2
A 6	73.2	11.3	70.2	8.5	10.8	−2.3

Fall in intraocular pressures as compared with fall in blood pressures from an artificially raised level. Group 1 and 2 have been carried out under identically the same conditions.

subconjunctival salt injection he showed that in this eye the percentage rise was less on compressing the aorta than in the normal eye, *i.e.*, the eye pressure goes up more when it is low to start with than when it is high at the start. This experiment does not give us the relationship we are looking for in that he has produced a wide dilatation of the intraocular blood vessels by irritation and so as the general blood pressure is raised it becomes increasingly more difficult for these vessels already dilatated to dilate further. We wish to find out then the following:

1. What is the form of the curve of intraocular pressure plotted against changes in blood pressure?

2. Does the form of this curve vary after the sympathetic has been cut? If, as the preceding experiments indicate, the sympathetic is effective in keeping changes of blood pressure from raising intraocular pressure it might be found that this protective action on the part of the sympathetic was more efficient the higher the blood pressure was raised.

EXPERIMENTS CLASS B

Methods:

We repeated the experiments as outlined under Class A with the exception that instead of sudden rapid rises and falls in blood pressure we arranged an apparatus for raising the pressure very slowly. We allowed four minutes in each case for the total rise, and made sure that the intraocular pressure did not lag behind. The blood pressure was raised slowly by fastening the ligature around the aorta to a block moving on a fine screw. Turning this screw drew out the ligature at a very slow rate and gave us a means for controlling the rise to any desired rate.

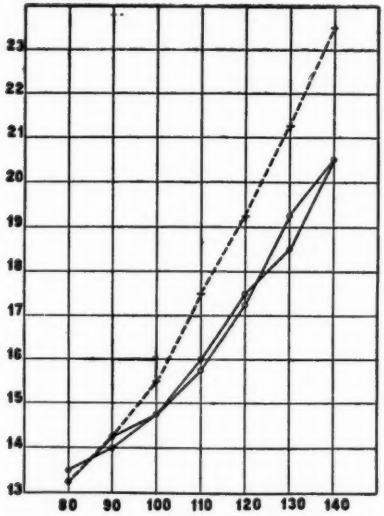
The following experiment is given illustrating the results obtained.

EXPERIMENT B–I

November 1, 1923—Cat, urethane, ether. Blood pressure left carotid. Intraocular pressure right eye. Both vagi cut in neck. Loose ligature around aorta. The blood pressure was raised and lowered twice, taking four minutes to each rise. The right sympathetic was then cut and the blood pressure raised two more times. Readings were taken of the intraocular pressure for every 10*mm* rise of blood pressure.

Before Section		After Section	
Blood Pressure	*Intraocular Pressure*	*Blood Pressure*	*Intraocular Pressure*
80*mm* Hg	13.25*mm* Hg	80*mm* Hg	13.25*mm* Hg
90	14.25	90	14.25
100	14.75	100	15.5
110	15.75	110	17.5
120	17.25	120	19.25
130	19.25	130	21.25
140	20.5	140	13.5
80	13.5	80	12
90	14	90	12.5
100	14.75	100	13.75
110	16	110	15.5
120	17.5	120	18.5
130	18.5	130	20.5
140	20.5	140	.....

The following curve has been made up from these tables. It will be seen that after a preliminary curved slope upward the relationship between intraocular and blood pressures is practically a linear function. After section of the nerve the intra-



Illustrating the protective action of the cervical sympathetic on keeping the intraocular pressure from rising the total amount due to rise of general blood pressure.

Abscissa = Intraocular Pressure in *mm* Hg.

Ordinate = Blood pressure in *mm* Hg.

The two unbroken lines are two separate readings with the sympathetic intact. The broken line is a reading on the same eye after the sympathetic was cut.

ocular pressure rises more than before section and by an increasing amount as the blood pressure is raised. This can be adequately explained on mechanical grounds, for after section of the nerve the blood vessels will continue to dilate still further as their internal pressure increases, effecting a greater and greater rise of intraocular pressure. It might be suggested

that as the blood pressure was raised higher and higher more powerful stimuli were sent out by the sympathetic to the ocular blood vessels, in an attempt to constrict them more and more.

In a recent paper of Byrne and Einthoven (10) they show an increased action current in the cervical sympathetic and a rise of blood pressure when a sensory nerve, as the sciatic, is stimulated. They regard the increased action current as a reflex from the sensory stimulus, and not as a result of the rise in blood pressure. In fact the increase in the action current precedes the rise in blood pressure by an appreciable interval. It would be extremely interesting and would bear directly on our results if it should be found that raising the blood pressure mechanically causes in itself an increased action current in the cervical sympathetic and this the greater as the blood pressure rose.

In 1902 Bayliss (11) described certain reactions of the denervated arterial wall to changes of internal pressure. He found that the muscular coat of the arteries reacts, like smooth muscle in other situations, to a stretching force by contraction. Von Anrep (12) later repeated Bayliss's work and while he obtained identical results his interpretation of them was different. He attributed his effects to an active constriction of the blood vessel wall by adrenalin which was poured into the blood stream from the adrenals when the blood pressure was raised. Although the controversy is not entirely settled in all the issues, it is clear that the peripheral arteries even when denervated act in such manner as to prevent their full dilatation to rises in internal pressure. Our experiments would show that in addition to this the innervation of the blood vessels by the sympathetic (in the eye at least) is a further protective mechanism.

SUMMARY.

When the blood pressure is raised in an animal the intraocular pressure is increased. If the rise in blood pressure be slow the rise in intraocular pressure is in direct proportion to it. When we compare the rise in intraocular pressure before and after section of the cervical sympathetic with the same

rise in blood pressure, we note that after cutting the nerve the intraocular pressure goes up higher, so that the sympathetic is effective in keeping sudden rises in general blood pressure from exerting their full effects on intraocular pressure. This gives experimental proof to the assumption of Magitot and others that there is a mechanism in the eye whereby changes in general blood pressure can be effectually regulated.

We find no effect on intraocular pressure of cutting the cervical sympathetic at normal blood pressures. By our instrument pressure changes of 0.25*mm* Hg can be registered, so that if there is any effect on the size of the blood vessels, it is too slight to affect intraocular pressure. On theoretical grounds, however, comparing the innervation of the blood vessels of the eye and ear by the sympathetic we believe that there is most likely a slight dilatation of the ocular blood vessels on cutting the nerve, only much less than there is in the case of the ear.

The fact that after section of the nerve the rise in intraocular pressure is greater the higher the blood pressure goes can be accounted for on mechanical grounds, and needs no further explanation. A possible hypothesis for which there is yet no experimental evidence is that, as the blood pressure ascends, stronger and stronger stimuli are sent out along the nerve to the blood vessels as a reflex.

CONCLUSIONS.

1. When the blood pressure is raised the intraocular pressure is increased. This increase, however, is kept in check by a local vaso-constriction of the ocular blood vessels through the cervical sympathetic. This mechanism is a protective one to the eye, and prevents sudden changes in general blood pressure from causing harmful changes in intraocular pressure.

2. This protective action is increasingly effective as the blood pressure ascends.

3. No effect on intraocular pressure is seen when the cervical sympathetic is cut at normal blood pressure.

4. In the normal animal as the blood pressure is raised the intraocular pressure rises in direct proportion to it.

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AN UNUSUAL DERMOID CYST

By Dr. HARRY S. GRADLE
(Attending Ophthalmologist Michael Reese Hospital)
and
Dr. JULES C. STEIN, Chicago.

THIS case is unusual in that it was brought for medical attention after being present seventeen years; there was a marked depression in the frontal bone underlying the tumor; pressure on the tumor would excite nausea and headache; and it presented other conditions very much like a cephalocele orbitæ anterior.

R. A., age 17, was admitted to the Michael Reese Hospital July 19, 1923, for a swelling in the right upper lid which according to his own statements, had been present since birth, gradually increasing in size. There has not been any pain but the upper lid has covered the eyeball and made it very uncomfortable. At times the patient would have headaches and if at anytime he would press on the mass in the lid he would have pains in the head as well as vertigo and often nausea.

There were no complaints relative to nervous, respiratory, cardio-vascular, gastro-intestinal or genito-urinary disturbances. Past history medically and surgically was negative and there was no history of any similar disturbances in the family. Venereal diseases were denied.

Physical examination revealed a healthy white male seventeen years of age. The shape of the head was normal and there were no gross anomalies to be seen. The pupils were regular and reacted normally to light and accommodation.

No nystagmus or strabismus. Nose and throat examination was negative. Chest, heart, and abdominal examination did not reveal anything unusual. Reflexes were normal.

In the right upper eyelid extending into the orbital tissue and subcutaneously onto the forehead is felt a rather movable semi-doughlike mass which cannot be compressed or reduced in size by manipulation. The mass is irregular, rounded in the portion in the lid but flattened as it is carried upon the forehead. It is freely movable in the subcutaneous tissues but cannot be manipulated sufficiently to map out the complete size and form posteriorly. Passing the finger along the orbital ridge there is felt a marked bony depression (defect ?) beginning about 1*cm* temporal to the foramen supraorbitale, which can be followed only a certain distance because of the presence of the mass. Whether this ends in an opening through the frontal bone or is merely a depression cannot be determined. On the nasal side a similar depression in the bone is noted at about the inner canthus which cannot be delimited. Pressure on the mass towards the frontal bone causes a feeling of dull vertex headache, while continued and increased pressure is decidedly painful and nauseating. There was no displacement of the eyeball.

Repeated examinations of the urine were negative as was the Wassermann reaction.

Neurological Examination.—Dr. Pollock.

Gait is normal. No weakness or ataxia of the upper or lower extremities. The Achilles jerks are normal and equal. The knee jerks are normal. The deep reflexes of the upper extremities are brisk and equal. There is no nystagmus or extra ocular plasy. Convergence is moderately good. There is no clumsiness of hands, no adiodocknokinesis, no obvious sensory defect, no asteriognosis, no apraxia. The planter, cremasteric and abdominal reflexes are normal. There is no objective evidence of involvement of the frontal lobe.

Neuro-Surgical Examination.—Dr. Davenport.

Examination reveals a defect in the bone as a result of the tumor. X-ray does not reveal any defect in the skull.

Ear Examination.—Dr. Sonnenschein.

No spontaneous nystagmus, no past pointing, no Romberg.

10 turns to right—horizontal nystagmus to left 18 seconds.
10 turns to left—horizontal nystagmus to right 18 seconds.
10 turns to right—past points to left.
10 turns to left—past points to right.

July 21st spinal puncture did not reveal an increase in pressure even with pressure on the tumor mass. Operated July 25th. General anæsthesia. Usual sterile preparations. Right eyebrow shaved. Incision along the eyebrow for 6*cm*. Subcutaneous dissection exposing tumor mass which was adherent to the subcutaneous tissues but freely movable with it. By blunt dissection the tumor mass was separated from the soft tissues of the lid and from the orbital tissues as well as far on to the forehead. With the tumor mass removed there was left a depression in the frontal bone irregular in shape and depth corresponding to the bed of the tumor. It formed an irregular saucer-like depression which gave a decided notching to the frontal orbital ridge with a valley between for the reception of the mass. Examination of the tumor revealed a unilocular cyst with a tough covering which contained a yellowish sebiform mass cheesy in consistency with some fine hairs within. The entire mass was about the size of a large walnut.

Histological Report.—The tissue was 3.5 × 1 × 1*cm* rather firm and with a small central cavity. Microscopical examination revealed a wall essentially the structure of external skin, with a substratum of connective tissue supporting an epithelial lining like that of the external skin with some sebaceous and sweat glands. There was no evidence of malignancy.

The condition presented some difficulties in diagnosis especially in eliminating a hernia cerebri (cephalocele). A depression in the bone was present but the base of it could not be palpated, nor determined with X-ray, therefore it was impossible to state whether there was a communication with the interior of the cranium. Pressure on the mass produced headache and nausea which is a symptom typical of a cephalocele. The only feasible explanation for this symptom is that the frontal bone is very thin where the tumor was found and due to this thinness pressure on the mass was extended directly to the cerebral fluid and cranial contents.

The depression in the bone was apparently present because of the pressure produced by the cyst which had been present since birth causing the abnormal development of the structures surrounding itself.

NODULAR IRITIS DUE TO THE BACILLUS OF HANSEN (LEPROSY). SLITLAMP STUDIES.[1]

By Dr. LUTHER C. PETER, Philadelphia.

THE case is one of unusual interest: first, because leprosy is rarely seen in this country; and second, because, by some, the iris is said to escape in leprosy, while according to others, an iridocyclitis is found in a limited number of cases. For the privilege of reporting the case, the author is indebted to Drs. Sigmund S. Greenbaum and J. F. Shamburg, by whom the case was referred for an eye examination.

The patient is fifty-three years of age, of Jewish birth, a resident of this country for twenty years. She contracted nodular leprosy about four years ago. She is the mother of three children, one living. Neither child nor husband has contracted the disease.

The skin surfaces are covered with leprosy nodules, and lesions have been observed in the mucous membrane of the nose and mouth. Both eyes are involved, although the patient has not been conscious of any disturbance in the right eye, and has experienced little pain and only a moderate amount of redness in the left eye. Curiously enough, there are no nodules on the skin of the eyelids, on the conjunctiva nor at the limbus,—the usual favorite seats of eye complications in leprosy. Studies by means of the loupe and oblique illumination reveal a fairly normal cornea in the right eye, absence of pericorneal redness, an irregular pupil due to two points of posterior synechia, and a light brown iris of fairly normal appearance with a few pin-point white dots in the iris stroma.

In the left eye, there is slight pericorneal redness, and a lusterless cornea with some clouding due to diffusely scat-

[1] Read before Section Ophthal., College of Physicians, Philadelphia, December 20, 1923.

tered droplike deposits on its posterior surface. Through the cloudy cornea, numerous irregularly-shaped white plaques, varying in size from a pin-point to areas almost as large as the pupil, are found scattered over the iris tissue, for the most part between the pupillary and peripheral borders, with several on the pupillary margin. The iris tissue is smooth and atrophic, and in some places depigmented. The pupillary border is bound to the lens capsule by annular synechia, and the small pupillary space is almost filled with dark brown pigment and exudate.

Fundus details are not obtainable in either eye because of the smallness of the pupils and the posterior synechia.

Corneal microscope and slitlamp studies are as follows:

Notwithstanding the almost negative findings in the right eye by means of the loupe and oblique illumination—other than posterior synechia and a few white pin-point dots on the iris,—the sliplamp studies are quite positive. The anterior corneal surface, under a magnification of twenty-four diometers, presents numerous point-like irregularities and opacities. The substantia propria presents a grayish appearance throughout its thickness. A limited number of "mutton-fat clumps" are visible on the endothelial surface. The aqueous is clouded by fine dustlike bodies. The circulation of the fluid in the anterior chamber is plainly visible and fairly rapid, indicating, as Von Der Heydt and others have pointed out, a receding rather than an active process. When the inflammation is at its height, the surcharged aqueous tends to slow down in movement when observed through a sufficiently clear media. The value of this phenomenon, in differentiating between an active and receding process, is definite.

The pupil is irregular in shape due to two points of posterior synechia. The iris is light brown in color, somewhat smoothed out, and in spots atrophic. On the iris tissue, between the greater and lesser vascular circles, a few fine pin-point dots, yellowish-white in color, are visible.

In the left eye, the changes visible by means of the corneal microscope and slitlamp illumination are pronounced and of unusual character. There is a moderate increase in size and number of the pericorneal vascular loupes; but newly-formed vessels in the corneal tissue are not observed. Like the cornea of the right eye, the anterior epithelial surface is irregular and studded with minute superficial opacities. The substantia propria is even grayer and denser than the right eye. The corneal nerves show no changes either in thickness or in termination. The most characteristic corneal pathology is found on the endothelial surface. Over the entire endothelial lining of Descemet's membrane are

closely studded "mutton-fat clumps," yellowish-white in color, the yellow hue being the result of the reflection of the light from the yellowish-brown iris. These clumps are round in shape, varying in size from minute pin-point spots to others of five and six times the size.

The entire posterior surface is lined with these droplets from limbus to limbus above and below and laterally. Contrary to the ordinary form of Descemetitis, the droplets do not tend to accumulate, the larger ones predominating towards the lower part of the pyramid, but large and small clumps are equally distributed over the entire endothelial surface. When sharply focussed, the endothelial cells stand out as a beautifully gilded mosaic, hexagonal in design.

It is difficult to obtain a sharp focus on the retina because of the clouded cornea, excepting in chance areas between the deposits on the endothelium. The pupil is almost completely occluded and secluded, the pupillary area being almost filled with dark brown pigment and exudate. The iris tissue is smoothed out and atrophic. Crypts are almost invisible and the iris stroma present an even homogeneous surface, to all appearance devoid of structure. Scattered irregularly between the greater and lesser vascular circles are yellowish-white patches varying in size from a mere pin-point, to areas one fifth the size of the pupil. Some of these patches are flat and apparently even with the surface of the iris tissue. These are dirtier in color and probably represent an older formation than others which are elevated, like banks of snow dotted with points of intense whiteness. Two of these areas are found on the pupillary margin, but none are observed in the periphery.

Phases of the case which are of special interest are:

1. The absence of invasion of the skin of the lids and the conjunctiva. According to Elliot (*Tropical Ophthalmology*), these are favorite seats upon which nodules of leprosy are apt to be seen. Lopez (*Arch. Ophth.*, Leipzig, Bd. xviii., S. 404) observed in his studies in Havana that the eye, including the lids and conjunctiva, was invaded in every case of generalized leprosy. Most writers on the subject agree that the lesions are usually found in the anterior segment of the eye.

2. Absence of pain and ciliary redness, with involvement of the ciliary body. On this point, Yudkin, in his review of the literature on ocular manifestations of leprosy, *A. Jour. of Oph.*, May, 1918, p. 303, says: "the ciliary body is often in-

volved, but it is rare to have on this account any acute symptoms, the only sign of cyclitis being deposits of exudate on the posterior corneal surface." Our experience in this case is entirely in accord with the thought expressed by Yudkin. In cases of iridocyclitis, from causes which are usually encountered, pain and ciliary redness are prominent symptoms of ciliary involvement when, in addition, "mutton-fat deposits" are abundant on the endothelial corneal surface.

3. The slitlamp findings are of special interest. The slitlamp and corneal microscope do not disclose anything which has not been grossly reported in the literature, but furnish a minute analysis of these characteristics which is not possible with either ophthalmoscope or loupe.

In the case reported, the superficial punctate keratitis could not be detected as such by means of the ophthalmoscope or loupe. That it is definitely present is easily determined by slitlamp illumination and corneal microscope. In addition, the graying and stippling of the substantia propria is evidence of the tendency of the superficial process to penetrate to the deeper layers of the cornea. The absence of newly-formed blood vessels in the cornea is explained by the absence of pronounced ciliary inflammation.

The character, intensity and distribution of the Descemetitis, in the absence of the usually accompanying pain and ciliary redness, is unusual. The general tendency of the deposits of exudate to be distributed in a more or less pyramidal shape, with the larger deposits below, is easily understood by observing the currents of the nutrient fluid in the anterior chamber, especially when the aqueous is surcharged with leucocytes and inflammatory exudate. Why, however, should these deposits be so equally distributed in the case herein reported? Von Der Heydt has offered the suggestion that possibly these exudates are thrown off in such abundance during sleep that the even distribution is brought about. The writer has no other explanation to offer.

The character of the nodules on the iris is open to discussion. Much difference of opinion exists as to the character of the areas observed on the iris and even as to the presence of iridocyclitis in leprosy. So far as the presence of iritis is concerned, there should be no reason for difference. Several ob-

servers have evidently not encountered or recognized it in the cases which came under their observation. On the other hand, others have observed it so frequently that the possibility of its existence can not be questioned.

The exact character of the nodules is *sub judice.* Lie, quoted by Elliot, denies that "isolated leprotic nodules ever occur in this membrane, but believes that there is a general infiltration of it with leucocytes and bacilli, the infection spreading from the ciliary body." Others believe that the areas found in the iris are definite leprosy nodules. Here again there is a pronounced difference of opinion as to the part of the iris involved by the placques or nodules. Yudkin, in his résumé of the literature, says: "the nodule may occur in any part of the iris, but probably the most favorable place is at the iris angle." Espada (Third Pan-American Med. Cong., Havana, Feb., 1901) found nodules in the irides of half the number of the cases studied by him. He discussed the type herein recorded, and found them especially abundant in the neighborhood of the sphincter.

In the case reported, none were observed in the periphery, and but a few at the pupillary margin. Many are so small that they escape detection unless the corneal microscope is employed. This difficulty of observation by ordinary methods may account in a measure for the difference in opinions expressed.

Shall they be regarded as nodules or as exudate,—leucocytes and bacilli, as Lie has suggested? They differ materially from the tuberculous nodule and nodules in gummatous iritis. From a most careful study with the slitlamp, the appearance is that of an exudate rather than a nodule. Some are flattened, probably old, while others are elevated, resembling clumps of snow with an irregular surface and studded with pin-point areas of greater whiteness. The latter in all probability are of recent formation. It is probable, nevertheless, that they do represent nodules similar to those seen on the skin surfaces, but as pointed out by Lie, consist of exudate, bacilli and leucocytes, with the latter predominating. There is no increase of pigment surrounding the plaques. The depigmentation is general throughout. It is not at all likely, however, that the plaques, even though they may be regarded as exu-

date, originate as the "mutton-fat clumps" on the posterior surface of the cornea from the circulation of the aqueous humor. If this were so and these areas enlarge and are formed by deposits from the aqueous, their distribution would be more symmetric. The entire iris surface should be studded with droplets of varying size. This is not the case in the patient herein reported. The slitlamp illumination is quite adequate to pick up even minutest points of such deposits if present. It is probable that the bacilli carried by the blood find activity possible in the iris tissue and that each plaque springs from a nidus of bacillary activity and that in this sense each plaque represents a nodule formation similar to that found on the skin, but modified by the tissue in which they develop. In the receding stage, as shown in the right eye, the areas flatten, shrink, and become more or less pigmented or discolored, and are difficult to differentiate from the surrounding iris. These receding stages probably indicate bacillary death and final absorption of the process. These same plaques, apparently in a state of recession, are present in the left eye in the midst of an advancing process—another fact pointing to bacillary activity as a starting point for each area of exudate.

Inasmuch as nodules are found on mucous surfaces, in the mouth and nares, differing materially in appearance from the nodules on the skin, but nevertheless nodules; so these exudates, found on the iris, may be called nodules or at least represent the same sort of process as found in skin lepromata. "Nodular iritis of leprosy," in the writer's judgment, is a correct phrase to indicate the type of iritis usually observed in leprosy. It is descriptive of a variety of iritis which is totally different from other forms of plastic and serous iritis.

FURTHER NOTE ON OCULAR SPOROTRICHOSIS.

By Dr. S. R. GIFFORD.

(From the Department of Ophthalmology, University of Nebraska Medical College, Omaha, Nebr.)

IN the *Archives* for November, 1922, I reported the case of a boy with an infectious process evidently starting in the lacrimal sac, which had destroyed its bony wall back into the ethmoidal cells. Sporothrix Schenkii was grown from the lesion in pure culture. Less than a year later, a second case of ocular sporotrichosis appeared, which presented a somewhat similar location of the lesion.

Helen W——, aged 19, living in a small town near Omaha, came in complaining of a sore on the left lower lid. It was first noticed a month ago appearing spontaneously like a pimple, but this had ulcerated at the tip, and the redness had been increasing in extent ever since. She presented a raised, reddened, lesion of the skin extending from the region of the tear sac about one third of the distance across the lower lid, being about 2 centimeters in diameter. The inner half showed a reddish, roughened granulation tissue, denuded of epithelium, with some thin purulent discharge partly covering it, coming apparently from a deeper pocket or fistula at the inner angle of the lesion while the outer half was covered by reddened epithelium. The preauricular glands were tender and slightly swollen. The tear passages were permeable and apparently not affected. Cultures were made, but as Dr. Harold Gifford thought the clinical picture characteristic of sporotrichosis, she was instructed through her family physician to take large doses of potassium iodide without waiting for the report. A zinc oxide dressing was applied locally. After five days, both cultures showed five or six colonies, which developed the dry, mammellated appearance characteristic of Sporothrix colonies. Smears of these showed the typical branching

threads, with spores budding laterally from the branches near their ends.

The patient was not seen for three weeks, having been taking K.I. somewhat irregularly, and not much change was noted in her condition. The infiltration, however, had not spread. Four minims of a 7% solution of K.I. were injected around the lesion, with 2% cocain (this treatment proving, by the way, quite painful). The lesions were soaked thoroughly in Lugol's solution, Lugol's solution was prescribed, with instructions to soak the lesions twice daily and afterwards apply a 7% K.I. ointment and the doses of K.I. were increased to 40 minims three times a day. Two weeks later the swelling of the glands was practically all gone, and the ulcerated area healed over, there being no more discharge from the deep fistula. The original area was still reddened and slightly raised. In another week the area was no longer raised and a month later only a slightly reddened area was left in the center of the original area. It was thought advisable, however, to continue small doses of the iodides for several weeks longer.

Serology:—At the patient's first appearance, her serum was reported by Dr. Michael Wohl as being negative to agglutination with his strain of Sporothrix. Serum taken at the second visit, however, gave in my hands, a positive agglutination with her own organism at 1:80, and with the organism obtained in our previous case at 1:40. This previous strain, which was reported as not producing pigment, was subsequently found to produce brownish pigment on glucose-containing media.

At the time of my first report, the fistula in the lacrimal region of our case was reported to be still draining. A recent letter from the parent states that this healed over soon after, and has remained so.

The location of the ulcerated area over the lacrimal sac, with the presence of a definite dacryocystitis in our first case, suggests the possibility that the organisms may relatively often find their original lodgement in the lacrimal sac, after having been blown into the eye with dust, and that they later grow in the walls of the sac or the surrounding tissue. In one of Morax's cases, the lacrimal apparatus was in good condition, but the location of the lesion over the sac suggested to

him the possibility that the organisms could have passed through the walls of the sac without infecting its cavity.

As I have seen no further reports of ocular sporotrichosis, since my last report, this case would seem to be the sixth American case, with a positive culture, to be reported.

ON THE VISIBILITY OF THE ACTUAL BLOOD STREAM WITH THE ORDINARY LOUPE.

By T. HARRISON BUTLER, Birmingham, England.

(With two figures in the text.)

IT is now a matter of common knowledge that the actual flow of blood in corneal and conjunctival vessels can be seen with the Czapski microscope and slit-lamp. It is probably not realized by many that the phenomenon can be perceived with an ordinary loupe giving a magnification of about eight diameters. The blood stream is visible with the corneal microscope when ocular F 55 and eyepiece 2 are employed, giving a magnification of nine diameters: there is therefore no inherent reason why it should not be obvious with the loupe which gives a similar enlargement.

To see the stream with the slit-lamp and microscope we manipulate the pencil of light in such wise that the blood-vessel is illuminated by transmitted light; if we wish to study the blood-stream in a corneal vessel we focus the ribbon of light furnished by the slit-lamp upon the iris, and view the vessel by light reflected back from the iris; by what Graves aptly calls *retro-illumination*. To see the flow of blood in a conjunctival vessel we allow the light to be reflected back from the sclera, and again make use of transmitted light.

The difficulty with the loupe and condensing lens is that, in the first place it is not easy to secure retro-illumination, and secondly that the low magnification limits the choice of vessels to those of medium size which generally have walls too thick to allow the movement to be seen under the somewhat imperfect retro-illumination at our command. In the case of the slit-lamp we have a more brilliant source of light, and the

Czapski microscope gives us the advantage of stereoscopic vision.

I first succeeded in seeing the movement in a case of strumous pannus. I found a vein of medium size extending one quarter of the way across the cornea. Using an ordinary focus-lamp I directed the light upon a yellowish iris, which is the best variety for retro-illumination, and accurately focusing the loupe I was first able to see that the column of blood in a branch was segmented. Careful study of the segmentation showed that the position of the segments altered gradually. I could not see the actual movement but in time the spaces changed places with the solid sections. It was like the movement of the minute hand of a watch, position altered although motion was invisible. Then as my eye became more dark adapted I noted that the blood column in the main vein had become moniliform, and finally quite suddenly I saw that the vein was full of streaming globules. I had before me the exact appearance that I see with the slit-lamp. I was able to demonstrate the movement to my house surgeon who told me the direction in which the blood was flowing. Figure 1 gives an

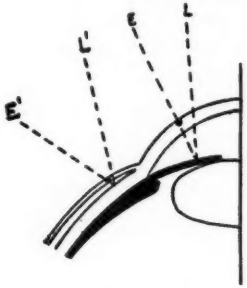

FIG. 1.

idea of the appearance. The slow stream in the side branches is marked with a minus arrow, the rapid stream in the main vessel with a plus arrow.

In this case the pannus was clearing up, and I imagine that the smaller vessel was becoming functionless and in consequence the stream was very slow.

I was unable to see any movement in other vessels in this

patient. The focus lamp and ordinary condensing lens proved inefficient, it was impossible to obtain a punctate focus.

With a 40 candle-power, gas-filled, half watt lamp I obtained better results, and better still with a 100 candle-power lamp at a distance. Here the source of light is smaller than with the focus-lamp and much brighter, and it was possible to obtain some approach to a punctate focus but it was surrounded by a disturbing area of diffused light.

A point o' light lamp solved the difficulty. This lamp furnishes a small bright spot of light which can be focused upon any desired spot, giving almost punctate illumination. If the light from the point o' light be carefully focused upon a limbal vessel in a normal eye and then moved slightly to one side it is possible so to arrange matters that the observing eye receives rays that have traversed the vessel. Under such circumstances if the eye be dark-adapted the blood stream will be perceived as it is in the corneal microscope. Spherical bodies are seen to be streaming along the vessel; they cannot be the actual blood corpuscles, for these are too small to be visible with a magnification of ten. The effect is probably an optical one due to turbulence in the blood stream caused by the unequal and variable caliber of the blood vessel. The intermittent stream which is seen with the slit-lamp in the capillaries has so far not been seen with the loupe. The magnification is not high enough to see motion in the smallest blood vessels.

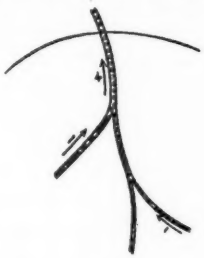

FIG. 2.

Figure 2 shows the conditions necessary to see the flow with the loupe or microscope. L L′ is the source of light, E E′ the observing eye.

The observation of the blood stream with the loupe is not a

mere curiosity. The technique necessary has a wider application. It is possible to some extent to apply slit-lamp methods to the simpler apparatus in general use. The beam from the point o' light traces out an illuminated path in the transparent media of the eye just as the ribbon from the slit-lamp does. We can see the cornea relucent; note that the aqueous is dark; and again that the lens is relucent. A sclerosing nucleus can be detected, and the depth of the anterior chamber estimated. The colorette of the iris can be examined by reflected lens light and holes and atrophic areas diagnosed. Deposits upon the back of the cornea can be examined in a rudimentary manner by retro-illumination, and if we employ specular light we can examine the lens shagreen and detect the sutures of the *adult* nucleus. (Unfortunately the *adult* nucleus has been translated from Vogt's work as the *senile* nucleus, and this has given rise to some confusion.) A further advantage in the method of focal illumination is that it is not as dazzling to the patient as the ordinary oblique illumination with diffused light.

Examination with loupe and condensing lens is certainly capable of further elaboration. I am having an aplanatic condensing lens made which will be corrected for spherical and chromatic aberrations, for such a lens will project a more perfect pencil of light than the ordinary simple lens, and it will cost about three dollars only. We cannot carry the slit-lamp about with us and many cannot afford to have one at all, but all can take advantage of any slit-lamp methods that are applicable to ordinary simple instruments. Using an aplanatic condensing lens which can be carried in a small case in the pocket, a loupe or a Zeiss prismatic single field glass, and a half-watt bulb, we can obtain some of the advantages of the slit-lamp. A point o' light lamp is not expensive and it never "lets you down." It is said that it will not run on an alternating current but this is a fallacy. I have used one for years as an operation lamp at the Coventry Hospital. There we have a 200 volt alternating current with 50 periods per second. It actually runs better than one I use with a direct current. The lamp should be housed in a simple box with a hole in it. The box should be on a ball and socket joint so that it can be adjusted as desired.

I am about to experiment with an inexpensive outfit containing a lamp of the nitra type with a short thick spiral filament. This will cost about $35, and will I hope be a step further in the search for a cheap method of focal illumination. The lamp will fit into the ordinary socket of the surgeon's electric bulb, and will be supplied with a suitable resistance to run from the town circuit.

ALLERGIC REACTION OF THE EYELIDS.

By Dr. EUGENE M. BLAKE, New Haven, Conn.

(Dept. of Ophthalmology, School of Medicine, Yale Univ.)

THE case to be described was unique in the experience of the writer and seems sufficiently unusual to warrant record. It bears upon the question of angioneurotic œdema which may, in reality, be a condition of allergy and not "spontaneous" as the older writers regarded it. It is in the hope that it may lead others to seek for the cause in cases of unexplained œdema of the lids, which we may find is an unnatural local reaction to foreign protein, that the case is reported.

In March of 1923, L. M., aged 22 years, a second year student in the School of Medicine, came to me with a very intense swelling of the right eyelids and surrounding parts, even down into the neck. The hands and arms were also involved. A half hour before the examination, while at work in the physiological laboratory on an experiment with a cat, blood had spurted into the right eye and onto the arms and hands. Almost immediately the parts became greatly swollen, itched severely and were covered with a faint-rough eruption. There was no difficulty in breathing except that the nose was partly occluded. There was moderate epiphora. The bulbar conjunctiva was slightly œdematous but the other ocular structures were normal and vision was unimpaired. Even in the short period between the accident and the patient reaching my office, the swelling had considerably decreased, so the patient stated, and the next day had entirely cleared. The only treatment recommended was the application of cold compresses.

The family history disclosed that the patient's father was a sufferer from a severe type of spring and autumnal hay fever.

In the patient's past history there was no instance of

spontaneous allergy. At the age of fourteen or fifteen he had diphtheria and was given 5,000 units of anti-toxin. He thinks that he had an urticaria the next day. At eighteen years of age he again had diphtheria and was given 20,000 units of anti-toxin within a half hour. There was no difficulty in breathing but his temperature rose rapidly and a general urticaria appeared later in the day. Five days after this the patient developed serum sickness involving all his joints and a temperature of 106° F. but without the occurrence of urticaria.

The patient had had the usual contact with domestic and laboratory animals without symptoms of any sort, but for a short period preceding the episode related, he had noticed a roughness and itching of the skin if licked by a cat or dog. On one occasion, a few weeks later, he had a recurrence of the œdema of the eyelids and nasal mucous membrane upon entering the laboratory but was subsequently able to handle a cat without symptoms.

Intracutaneous tests made a few weeks after the allergic reaction in the eyelids and hands, showed the patient sensitive to the following substances:

Sera		*Epithelia*	
Rabbit	1—5,000	Rabbit	(0.4 mg of N. Per *cc*)
Cat	1—1,000	Cat	(0.001 " " " " ")
Horse	1—100	Horse	(0.01 " " " " ")
Guinea Pig	1—10	Guinea Pig	(0.10 " " " " ")

The patient was not skin sensitive to more dilute solutions of the animal epithelia nor to goose, chicken, goat, or dog epithelia or ragweed in the usual test dilution. More concentrated solutions were not tried. He would undoubtedly have reacted to more dilute solutions of cat and horse serum had they been tried. We may say, therefore, that this individual was extremely sensitive to cat, horse, and rabbit sera and only difficultly so to guinea pig serum. He was much less sensitive to the hair of these animals, except the cat, but was very definitely so.

I wish to express my thanks to Dr. Ruth A. Guy, of the Department of Pediatrics, who made all the tests and supplied me with the history of the case. It is presented as an ophthalmological case because it bears on the question of œdema of the lids and because this was what most worried the patient.

OPERATION FOR CROSS-EYE WHERE GLASSES ALREADY CORRECT THE DEFORMITY. REPORT OF FOUR CASES.

By Dr. J. A. KEARNEY, New York.

FOUR patients have applied to me recently for the correction of cross-eye, all having glasses, which when worn, corrected the defect, but immediately upon the removal of the glasses, in each case one eye turned in (convergent concomitant strabismus).

The average young patient will wear glasses religiously that give relief of symptoms for occupational and even general use, but will nearly always set them aside when attending certain social functions in the younger set regardless of consequences. Those who use glasses to correct a cross-eye defect are forced to wear them at these functions, where as a rule, all of their young associates are without glasses and naturally they feel conspicuous. Sensitiveness on this account causes some of them to consult an eye surgeon for operation which will correct the deformity in order that they may eliminate the glasses at such times. The patients' point of view, while not corresponding to ours, seems vitally important to them and on this account our hands are sometimes moved to operate. It is remarkable that those who have consulted me whose cross-eye defect was already corrected by glasses were more solicitous and insistent for operation than cross-eyed patients whose defect could not be remedied by glasses. In the former instance we usually advise no surgical interference, while in the latter, we always advise operation when conditions are suitable after the usual non-operative measures have been tried and failed to give result.

With suitable conditions obtaining we employ the same

operative procedures for correcting cross-eye defects in the cases cited here as in other cases of cross-eye where glasses do not have any effect upon the deformity.

The glasses that are usually worn to correct a cross-eye deformity are of such strength that even after a successful operation they will still be found necessary for close work and in the majority of instances for general use.

The writer's usual determinations before advising an operation for concomitant convergent and concomitant divergent strabismus whether glasses do or do not correct the squint are the extent and character of the rotations of the deviating eye inwards and outwards.

The lateral rotations are sufficient if the external limbus of the cornea extends to the external canthus when the eye is rotated outwards as far as possible and the internal pupillary margin under the ordinary illumination is on line with the two lacrymal puncta when the eye is rotated inwards as far as possible and the character of these movements is prompt and agile.

In the convergent concomitant strabismus when lateral rotations of the deviating eye are sufficient a complete tenotomy of the internal rectus muscle of the affected eye usually corrects 15° of squint; and advancement operation upon the external rectus muscle of the deviating eye usually corrects 15° of squint; a complete tenotomy of the internal rectus of the deviating eye followed by an advancement of the external rectus muscle of the same eye as soon as the wound heals after the tenotomy of the internus usually corrects 30° of squint; a tenotomy of the internal rectus muscle of both eyes at the same time usually corrects 45° of squint. A complete tenotomy of the internal rectus of the deviating eye followed by an advancement of the external rectus of the same eye as soon as the wound heals after the tenotomy of the internus, followed by an advancement of the external rectus muscle of the non-deviating eye when healing has occurred after the advancement operation of the externus in the deviating eye usually corrects 45° of squint.

In divergent concomitant strabismus when lateral rotations of the deviating eye are sufficient a complete tenotomy of the external rectus muscle and an advancement of the in-

ternal rectus muscle of the deviating eye at the same time corrects squints up to 45° depending upon the placement of stitches in the internus rectus muscle.

A complete tenotomy of the internus rectus muscle means a complete section of the tendon *2mm* from its insertion and section of all aponeuroses of the muscle as well. A guarded mattress suture is always placed *2mm* distally to the point of section of the internus before the muscle is cut and allowed to remain in the cul de sac for five days as a precautionary measure should over correction or dysfunction of the internus happen after the operation.

The advancement operation upon the external rectus muscle of the deviating eye that has given the writer the best uniformly good results and which usually corrects 15° of inward deviation of the eye when the lateral excursions are of good character and normally sufficient in extent is as follows: A *10mm* vertical incision is made in the conjunctiva *6mm* outwards from the corneal limbus, *5mm* either side of a horizontal line that would bisect the cornea if extended. Above and below the margins of the muscle just beneath the conjunctival incision the tissues down to the sclera are grasped by a fine single toothed forceps and cut when raised, making button holes through which the point of the tenotomy hook enters the lower one, passes under the muscle and emerges in the upper one. The adherent conjunctiva is dissected from the surface of the muscle and the margins of the muscle are freed of aponeurotic bands as far back as possible. One of the blades of the advancement forceps is then passed beneath the muscle and at right angles to it *5mm* beyond its insertion and the forceps locked tightly. Tenotomy hook is removed. Two separate sutures of twisted black silk No. 3 are employed each on a separate needle. Traction is exerted upon the muscle forwards and inwards by the advancement forceps and one needle is carried around the upper margin beneath the muscle and passed from within outwards through the center of the muscular tissue *5mm* beyond the grasp of the advancement forceps. This stitch is repeated and forms a loop around the upper half of the muscle fibers—the so-called whip stitch. The suture is then passed through the conjunctiva lying over this part of the muscle from within outwards and

1*mm* back of the incised margin. With the other suture this whip stitch maneuver is duplicated in the lower half of the muscle and in the conjunctiva overlying it. When both stitches are found to be securely placed a section of the muscle is made 2*mm* from its insertion. The needle of the upper suture is entered deeply into the tissue of the stump at the insertion of the muscle 1*mm* below its upper border and the suture is pulled through it. The needle is then carried beneath the conjunctiva lying over the stump and emerges 1*mm* from the corneal limbus in the position of one-thirty o'clock on the cornea. The needle of the lower suture is entered similarly into the stump at the insertion of the muscle 1*mm* above its lower border and a similar reënforcement stitch is placed under the conjunctiva emerging 1*mm* from the corneal limbus in the position of four-thirty o'clock on the cornea. The two stitches are now ready for tying. Traction is exerted inwards upon the muscle by the advancement forceps which is still in place and only slight traction outwards is necessary upon the inner portion of the upper suture emerging from the conjunctiva near the cornea to place the tissues in position to be tied without exerting any traction upon the sutures in the muscle. A double ordinary knot is then tied which secures the upper suture. At this step in the operation the advancement forceps are released and the excess muscular tissue excised. The lower stitch requires very little traction and is tied similarly to the upper stitch. It will be found that the conjunctiva requires no stitches when both sutures are tied as its incised surfaces are opposed.

Concomitant squints in the majority of instances measure 15°, 30° and 45° and the lateral rotations of the deviating eye are usually sufficient. The management of variations in concomitant squints from sufficient character and extent of the lateral rotations and from the usual angles of deviation depends upon the judgment of the individual operator.

CASE 1.—F. de B. F., New York, aged 23, broker and member of the younger society set, comes because he would like to discard glasses when interviewing prospective clients and also during his social pastimes. His mother first noticed his left eye turn in when 3½ years of age. Glasses were prescribed at this time but they did not reduce the

squint. At 5½ years of age another eye surgeon tenotomized the internal rectus muscle of the right eye. This operation did not fully correct the defect but the eye became straight thereafter when he wore his glasses.

He consulted me May 18, 1920. At this time his eyes were straight when he had his glasses on but upon removal of them, the right eye turned in 15°—internal and external rotations of the squinting eye were sufficient and the internal rectus had been previously tenotomized. An advancement operation upon the right external rectus was suggested as a measure that would possibly correct the defect. This he accepted. On May 20, 1920, the writer's advancement operation upon the external rectus of the right eye was done and ten days afterwards stitches were removed. Eyes were perfectly straight then without glasses, also with glasses and have remained so ever since. Lateral rotations of the operated eye are sufficient and agile. Vision with correcting glasses: Right Eye—S plus 5.00 equals $\frac{20}{20}$; Left Eye—S plus 5.00 equals $\frac{20}{20}$.

Case 2.—A. C., New York, aged 23, living at home, whose right eye turns in upon removal of her glasses. Embarrassment she suffered while dancing when her nose-mounted glasses would slip and she did not want her fiancé to know her eye crossed when her glasses were removed—these were the compelling causes for consulting an eye surgeon for operation. Her mother noticed her eyes crossed when she was six years of age and at that time glasses were prescribed, correcting the defect and have done so ever since. Without glasses her squint measures 15°, right eye turning in. Lateral rotations of the squinting eye were sufficient. Spectacles instead of nose-mounted glasses were advised and were refused. A tenotomy of the right internal rectus muscle was then suggested as a measure that would possibly correct the defect. This she accepted and the operation was done the same day—September 15, 1920. After operation the eyes were straight without glasses and have remained so ever since. Lateral rotations of the operated eye are sufficient and agile. Vision with correcting glasses: Right eye S plus 4.50 combined with C plus 50 ax 90/ equals $\frac{20}{20}$. Left eye S plus 3.50 combined with C plus 50 ax 90/ equals $\frac{20}{20}$.

Case 3.—J. M., New York, aged 24, a neighbor of the previous patient, insisted upon an operation because of the success in her friend's case. Her mother noticed her eyes crossed when she was six years of age; glasses were prescribed then which corrected the squint and her eyes have

been straight ever since when glasses were worn. She was wearing nose-mounted glasses when she consulted me and upon removing them, the squint measured 15°—left eye turning in. Lateral rotations of the squinting eye were sufficient. Spectacles were advised to be worn continually instead of nose-mounted glasses and were refused. A tenotomy of the left internal rectus was done December 14, 1920, which corrected the defect without glasses and the eyes have remained straight ever since with and without glasses. Lateral rotations of the operated eye are sufficient and agile. Right eye: S plus 3.50 equals $\frac{2}{2}\frac{0}{0}$. Left eye: S plus 5.50 equals $\frac{2}{1}\frac{0}{0}$.

CASE 4.—A. S., New York, aged 23, stenographer, left eye turns in when her glasses are removed. She became engaged to marry the superintendent of the establishment in which she was employed and desired her eyes straightened without the aid of glasses as her fiancé was not aware that a deformity existed when her glasses were removed. Her mother noticed that her daughter's eyes crossed a short time after she entered public school and she was then about six years of age. Glasses were prescribed at this time that corrected the deviation. She has been wearing glasses ever since. Without glasses her squint measured 30°—left eye turning in. Lateral rotations of the squinting eye were sufficient. I advised her to wear spectacles instead of eye glasses and to keep them on continually, but she insisted upon operation. On September 25, 1923, the internal rectus muscle of the deviating eye was completely tenotomized which corrected 15° and there was 15° of deviation still left uncorrected. On October 4, 1923, after the tenotomy wound healed the external rectus muscle of the same eye was advanced. (Writer's advancement.) Eyes were straight after this operation without glasses and with glasses and have remained so to date. Lateral rotations of the operated eye are sufficient and agile. Vision with correcting glasses—Right eye: S plus 1.50 in combination with C plus 50 ax 90 equals $\frac{2}{2}\frac{0}{0}$. Left eye: S plus 1.50 in combination with C plus 50 ax 90 equals $\frac{2}{2}\frac{0}{0}$.

REFERENCE.

KEARNEY, J. A. "Correction of Cross-Eye Deformity." *American Journal of Ophthalmology*, Series 3, vol. ii., p. 124, 1919.

A CASE OF ENCAPSULATED ANGIOMA OF THE ORBIT.[1]

By Dr. W. GORDON M. BYERS, Montreal.

THE following case, of little surgical moment, is reported to help balance the picture of the encapsulated angiomata of the orbit, which largely, so far, has been filled in by records of the more menacing growths of this nature. Growing out of the case, too, are a few points of clinical and pathological interest.

The patient, a male aged 50 years, complained that a swelling had been gradually developing behind the right lower lid for about two years. The rate of development had not been entirely uniform; progress had been more rapid at certain times than at others. It was felt that the swelling was somewhat more pronounced when the patient was fatigued; but variations in size were not noticed in connection with stooping or conditions that favored congestion of the orbit. There was no pain.

On examination one saw the right lower lid puffed out 10 to 15*mms*, as if from œdema. The swelling began just below the margin of the lid and reached to the lower margin of the orbit, rounding up gently between these two points. It was somewhat more noticeable over the outer as compared with the inner half of the lid. On palpation one felt a tense, rounded mass between the globe and the margin of the orbit. This appeared to be adherent neither to the globe nor to the orbital walls, and could be shoved back easily into the orbital fat. It was quite free from tenderness. There was no apparent displacement of the globe, and the ocular movements were perfect. Possibly the tumor moved slightly in association with the upward movement of the eyeball. The fundus of the right eye was

[1] Read before the American Ophthalmogical Society, Colorado Springs, June 20, 1923.

normal, and vision equalled $\frac{6}{6}$ − with + 0.75 sphere ⊃ + 0.75 cyl., ax. 15°. The left eye was normal, and vision equalled $\frac{6}{6}$.

Under general anæsthesia, the tumor was removed without difficulty through a skin incision. It was quite globular in shape, and had a diameter of approximately 1*cm*, in its fresh state.

PATHOLOGICAL EXAMINATION.

Microscopically the growth presents all the characteristics of a "cavernous" angioma. A well-marked capsule entirely surrounds the tumor and walls it off from the orbital fat, remains of which, with widely dilated vessels, are seen here and there at the periphery of the section. Within the body of the growth are venous-like spaces of varying size. These are filled for the most part with red blood cells and a relatively large number of polymorphonuclear leucocytes that show a tendency to collect at the periphery. In other places the contained blood has undergone degeneration. Supporting the vessels are septa of fibrous connective tissue in varying stages of development. In places this connective tissue is decidedly embryonic in character; in others it is dense and void of nuclei. The trabeculæ are thickened between the smaller and more recently formed vessels, and thinner between those of larger size. The latter can be noted here and there to be in process of coalescence. Replacing the fibrous connective tissue, one sees at times, especially toward the periphery, masses of lipomatous and areclar tissue; but generally speaking the fibrous connective tissue predominates.

COMMENT.

Two points struck me in connection with this case. Arbitrarily dividing the orbit by a plane tangent to the posterior surface of the eyeball, I found on analysing a considerable number of records that the angiomata are more frequent in the posterior than in the anterior half of the space. The position of the present growth in the median line of the orbit below and in front was, therefore, exceptional. The other point was, that in the orbit with its rich system of connective tissue stromata, the connective tissue elements of the angiomata oc-

curring here (capsule and trabeculæ) must necessarily be a pronounced feature. In the lower anterior part of the orbit, where the present case grew, the septa are especially well developed.

There seemed to be an obvious connection in the case of these angiomata between their location and the vascular supply of the orbit, as well as between their histologic characteristics, and the distribution of the orbital connective tissue. My colleague, Dr. S. E. Whitnall, Professor of Anatomy at McGill University, to whom I submitted the questions, felt that the inferences were justified by a study of the general topography of the veins and connective tissue septa of the orbit; and he was kind enough to draw up for me the following very interesting summary of the anatomical findings in support of his statement:

"There are two main venous trunks: (a) The superior ophthalmic vein, the chief one, formed behind the medial corner of the upper eyelid by the union of two anastomoses with the supra-orbital and angular veins of the face. Its numerous tributaries are from the upper part of the eyeball (venæ vorticosæ and ciliary veins); from the upper medial ocular muscles; and the two ethmoidal veins. It also receives the lacrimal vein, which is often large enough to be considered as a third main trunk. (b) The inferior ophthalmic vein, the vein of the orbital floor. It receives tributaries from the lacrimal sac, conjunctiva of the lower eyelid, lower part of the eyeball, and lower and lateral ocular muscles. It anastomoses freely with the superior vein, and in most cases finally empties into it at the apex of the orbit. The termination posteriorly of these trunks into the cavernous sinus is usually by a single channel, though separate entry of the superior and inferior ophthalmic veins and central vein of the retina sometimes occurs.

"Now inspection of the orbit in the skeleton, together with a study of frozen sections with the contents entire, shows that the whole cavity may be divided into a roomy quadrilateral anterior half in which is lodged the eyeball, lacrimal gland, and lacrimal sac, and a narrower triangular posterior half, wherein lie chiefly the optic nerve and bellies of the ocular

muscles. It is in this posterior or retro-ocular region that most of the tributaries of the ophthalmic veins lie; for being situated behind the eyeball there are found here the numerous venæ vorticosæ, muscular, ethmoidal, and lacrimal veins, passing backwards to their terminations in the main ophthalmic stems. Towards the extreme apex of the orbit, all these vessels have, as noted, usually entered one stem, the superior ophthalmic vein.

"The result of this disposition of the vessels is, that if we imagine the orbital cavity to be divided by frontal planes into four regions of equal depth, of which the eyeball occupies the anterior two, then the vessels will be found in their most numerous distribution in the third quadrant, immediately behind the eyeball.

"On the other hand is to be noted that it is in this same retro-ocular quadrant that the orbital fat, through which the veins run, is most loosely packed and yielding, and exhibits in dissection least connective stroma; since here the optic nerve makes its widest excursions in harmony with the oscillations of the eyeball, and the muscle bellies presumably exhibit their greatest range of expansion, being tendinous at their two extremities. At the apex of the orbit (posterior quadrant) the orbital fat is greatly reduced in quantity, the space being compactly fitted by the origins of the muscles, nerves, etc.; but round the equatorial region of the eyeball that fat is enmeshed in far denser connective tissue."

REPORT OF THE TRANSACTIONS OF THE OPHTHALMOLOGICAL SECTION OF THE ROYAL SOCIETY OF MEDICINE.

By Mr. H. DICKINSON, London.

A meeting of the Section was held on January 11th, Mr. A. L. Whitehead, President of the Section, occupying the chair.

A Case of Late Sympathetic Ophthalmia.

Col. H. Herbert, I.M.S. (Retired), read a contribution on this subject.

He said the disease followed a trephining operation, performed without iridectomy, for **congestive glaucoma,** in November, 1917. More than five years elapsed before the other eye became involved. At the time of the operation, the patient, a woman, was aged 62. The operation did not entirely relieve the tension, and the congestion persisted for more than a month. After that, it gave no more trouble until the fellow eye was attacked, and then injection and tenderness returned. In the sympathizing eye the disease was comparatively mild, and seemed to yield completely to three months of simple mercurial treatment.

In this case there was ample histological evidence that the operated eye, during the five quiet years, was only relatively quiet. The notes mentioned only a dense white pupillary opacity, and the fact that the eye was blind. There was good reason for believing the eye was the seat of a very feebly progressive infective process throughout.

The nodular character of the uveitis was very noticeable, the foci of greatest infiltration standing out as round white

points in the iris, ciliary body and choroid. He described the cytology by means of a number of slides.

As a probable indication that the unknown specific organism had become acclimatized outside the uvea, large-celled infiltration could be followed through the tissues filling the trephine hole, and through the sclera, along the anterior perforating blood vessels above and below the cornea. It had spread into the conjunctiva, and backwards immediately superficial to the sclera. There was another extra-uveal site of activity beneath the thick pupillary membrane noticed clinically between the membrane and a thick capsular cataract. The detached retina was folded and thickened, and the ciliary body and choroid were more or less detached from the sclera, and their layers were widely separated. The inner surface of the choroid below merged into a thick layer of new fibrous tissue, containing masses of pigment granules, derived possibly from the retinal pigment layer; pigment had also wandered into the detached retina.

With regard to the bearing of the case on the operative treatment of glaucoma, he had not heard of any instance of the disease following an intentional iris-inclusion operation for over twenty years, and he believed a correctly performed iris-inclusion operation was quite free from this risk; but he held that correct performance included effective conjunctival antisepsis. In the present case the trouble was probably due to the lack of this.

The PRESIDENT said he thought most members would disagree with the statement that in most cases of this operation there was some incarceration of the iris.

Mr. PICKARD said he had had two or three cases of chronic glaucoma affecting both eyes, in which suddenly, for no obvious reason, one eye developed a condition with severe irido-cyclitis leading to blindness, and its clinical appearance would be taken for that of sympathetic ophthalmia. In some cases of chronic glaucoma he thought the glaucoma was the expression of some very mild cyclitis without the ordinary symptoms of irido-cyclitis. He had notes of cases with clinical symptoms of sympathetic ophthalmia though there had been no operation on the other eye.

Sir WILLIAM LISTER remarked that the question of the in-

carceration of iris in the trephine operation struck hard at the basis of technique. It was very important to avoid any iris incarceration whatever. Cases of late infection he considered to have been associated with the entanglement of some iris in the wound. Directly the knuckle of iris appeared through the trephine hole, it was very important to take hold of the posterior portion of the knuckle and pull it up, pulling out sufficient iris so that when the cut was made the iris spread right away from the opening. Where the pupil was large, the operator should aim at doing a complete iridectomy right through to the pupillary margin, so as to avoid incarceration.

M. E. TREACHER COLLINS suggested that unless Col. Herbert made serial sections through the complete trephine hole, he could not be sure there was no entanglement of iris in this case. In this case it was open to question whether it was not a late infection. In one of the sections shown there was infiltration of the conjunctiva round the trephine hole, and that might have been the source of infection.

Mr. R. AFFLECK GREEVES suggested that one of the most important things in trephining was to split the cornea forwards; sometimes the trephine was put too far back, and then incarceration of iris was likely to take place. He had cut sections of a series of eyes which were trephine failures, and in each case the failure was due to incarceration of the iris, caused by the trephine having been put too far back.

M. LINDSAY REA related a case in which he had to trephine two eyes in an urgent case of glaucoma, and the iris in one was incarcerated round the edge of the trephine hole, and he was unable to free it. As soon as he replaced the flap, he instilled atropine into the eye, and kept it under its influence with the result that in less than a fortnight the iris pulled well away from the trephine hole, and the eye did well, vision being restored to $\frac{6}{9}$ partly.

The PRESIDENT agreed with Sir William Lister as to the importance of securing freedom of the iris from the wound; he would do a complete iridectomy rather than leave any trace of iris in the trephine opening.

Col. HERBERT replied on the discussion. He agreed it might not be quite fair to say the average trephine hole contained some iris, and in that respect he might have too readily

accepted Elliot's statement. Probably the slight iris incarceration in this case had something to do with the disease. He did not doubt in his own mind that it was sympathetic ophthalmia, and the histology he regarded as characteristic, as revealed by the slides he had shown. In answer to Mr. Collins the tissue on either side of the trephine hole was serially examined. The infiltration of the conjunctiva was exactly like that of the uvea.

The Practical Value of the Slit-lamp.

Mr. T. HARRISON BUTLER read a paper on this subject, and gave a demonstration of the lamp. His paper was illustrated by a series of epidiascopic representations.

He said many still had the idea that the slit-lamp was useful mainly for research, that its function was chiefly anatomical. Alvar Gullstrand had opened up an epoch in the specialty comparable to that produced by Babbage's discovery of the ophthalmoscope. Probably as much had been learned from a study of the fundus and media as was possible by present methods but the slit-lamp enabled the problems to be attacked afresh, not only by supplying a vastly increased magnification, but by permitting the illumination of structures from behind, and, above all, enabling the anatomy of the eye to be seen in optical section. Slit-lamp technique could be to some extent applied to ordinary oblique illumination with condensing lamp and loupe, and when the observer became accustomed to the aspect of the cornea, iris and lens under high magnification, he was the better able to interpret the same subject seen with the smaller magnification of the loupe. By means of a 40-candle, half-watt gas-filled lamp, a ray of light could be made to pass into the eye, and the cornea could be seen to be illuminated, the anterior chamber dark, and, in cases of lens sclerosis, that there were dissociation areas in the lens. If the patient fixed his gaze between the light and the lens, the lens shagreen and the sutures of the senile nucleus could be seen, also the position of opacities in and under the capsule. By lens light one could see holes and atrophic areas in the iris collarette. With this lamp he had seen the circulation in the vessels at the limbus of a normal eye; the light was

reflected from the sclera, and traversed the vessel, thus fulfilling the conditions necessary to see the circulation.

He did not claim to possess expert knowledge of the slit-lamp, though he had taken Professor Vogt's course at Zürich, and had been working with one in Coventry Hospital for four months. During that time he had learned more about the anatomy and pathology of the exterior segment of the eye than with the older methods he had obtained in four years. It was essential to have the correct instrument, namely, the combined slit-lamp and Gullstrand simplified ophthalmoscope. He described the details of the instrument, and the functions of the various parts.

The examination must be made in complete darkness; the more difficult objects would be seen only when the eye was dark-adapted. It was very important that the microscope should be carefully adjusted for inter-pupillary distance, as the slightest error in this respect disturbed the stereoscopic effect, and caused eye-strain. Little or no discomfort to the patient was occasioned by the examination, but cases of interstitial keratitis were unsuitable in this respect.

Instruction in the use of this lamp was quite as necessary as in the use of the ophthalmoscope; practice should commence on pigs' eyes, and be continued on the normal human eye. Koeppe's valuable work on *Microscopy of the Living Eye* was an absolutely necessary work to be studied from the microscopical side of the question.

Mr. BUTLER emphasized the fact that the slit-lamp does not supersede present methods, but rather amplified and supplemented them. It enabled much which formerly was problematical to be clear. When the ribbon of light was focussed on the cornea, the illuminated part of the structure could be regarded as a prismatic block of light, and this enabled the observer to know, with reasonable accuracy, whether an object or structure was on the corneal surface, within the corneal tissue, or on the endothelial surface. By narrowing down the slit, one could more or less abolish the corneal and endothelial surfaces, and obtain a pure section. He exhibited slides showing the effect produced by a corneal ulcer, and how fluorescine penetrated deeply into the stroma. A conical cornea gave a prism whose anterior surface was more strongly curved than

the posterior. He had found the slit-lamp of service in the case of foreign bodies embedded deeply in the cornea; with the knowledge so obtained one could plan the method of extraction, and do the extraction with the least damage to the cornea. But it was in inflammatory conditions of the eye that the examination of the cornea was of special value. The endothelium of the peripheral areas of the cornea gave the appearance of being covered with small drops, like the mirror in a hot bath-room, a process Vogt called "bedewing"; that authority believed it to be due to a change in the endothelial cells. Koeppe, however, considered that it was caused by a cellular deposit. A case of interstitial keratitis could not be completely interpreted without the slit-lamp; with it, the extent and position of the vascularization could be mapped out. Symmetrical radial folds in Descemet's membrane and Bowman's membrane, had a diagnostic value in the case of a boy with irido-cyclitis, figures of which were shown.

Observation of the aqueous was very difficult, and called for complete dark adaptation. In a recent case of iritis he noticed that the aqueous was relucent. Careful study revealed flocculent gelatinous masses, and the presence of these was generally supposed to denote a gonococcal origin. The presence of cells in the aqueous was the first sign of irido-cyclitis, and was especially valuable in the case of a sympathizing eye. The presence of these cells could be held to be an early warning of sympathetic ophthalmitis.

The microscopy of the iris gave merely an enlargement of what was seen with the loupe.

The slit-lamp was of most value in connection with the lens. The optical section given by a narrow ribbon of light enabled the lens to be seen in profile, exactly as though it had been cut with a knife. Vogt differentiated the following regions: the capsule, the cortex, the senile nucleus, and the embryonic nucleus. There was a dark layer immediately under the capsule, whose significance had not yet been explained. Clinically, the lens could be regarded as three lenses, one within the other, and these gave mirror effects to focal illumination. The recognition of these facts greatly enhanced the powers previously possessed of diagnosis and prognosis, and also had a definite influence on treatment. He noted in a

case of diabetic cataract that the lens capsule was greatly thickened; and in such a case, if he had to operate, he would use the capsule forceps in preference to the capsulotome. Barraquer made a careful slit-lamp examination of every lens before using his suction apparatus on it; by this means he could form an opinion of the conditions he would meet. Mr. Butler showed four figures illustrating a case of combined cortical cataract and nuclear sclerosis. Examination of the lens in mirror light showed that the whole surface was covered with nipple-like eminences, apparently on the same level as the shagreen. In section this was seen not to be the case; the elevations were on the surface of the senile nucleus. This was seen to be highly sclerosed, and very yellow. The contour of the nucleus was much more curved than that of the lens itself. When the internal lens became sclerosed and more highly refractive than the cortex, the stronger curvature came into play and caused lenticular myopia, which, in this case, was 5D.

One of the chief uses of the slit-lamp was the power it conferred of recognizing slight signs of cyclitis. He carefully investigated every case of cataract, so that if the case should subsequently come to extraction, he had knowledge of the nature of the changes in the lens.

So far, he had not found much help from the lamp in the examination of the vitreous. It called for Koeppe's mirror and Vogt's micro-arc lamp.

The chief drawback in regard to the use of this instrument was the extra time required in the examination of cases, in his own clinic it was an additional 50%. But the extra time was well spent, as it added enormously to the interest and accuracy of eye work. The method was sufficiently specialized to warrant the formation of a small Society of enthusiasts, to compare notes and instruct and be mutually helpful.

Mr. BASIL GRAVES said that some time ago he heard Mr. Treacher Collins express the view that it was likely the slit-lamp was destined to have on ophthalmology an influence, in all probability, comparable to that for which many years ago the introduction of the ophthalmoscope was responsible. The slit-lamp and binocular microscope were of the greatest service in daily routine clinical work; they permitted a case to be

examined with greater ease and confidence, and with the expenditure of less time and energy, for the every-day pathological features for which it might be customary to search more laboriously by means of the monocular loupe with ordinary illumination. Certain important clinical features could only be seen or located by this new apparatus. Dr. Vogt's statement that the facility afforded by the slit-lamp enabled the prodromal manifestations of sympathizing ophthalmitis at a far earlier stage than heretofore, was alone a justification for possessing the instrument.

With regard to its use in training students in ophthalmology, its use in detecting and recording many features gave ample scope in training accurate observation and scientific deduction.

In reference to the clinical aspect, Mr. Graves said that in the present stage of development the method of pursuing work of this nature fell under the choice of three main headings. Firstly, a general slit-lamp examination of any patient whose eyes were normal, or of other patients irrespective of the eye condition from which they were known to be suffering. In examining any old woman's eyes with a binocular microscope and slit-lamp one had a museum before one. As Lang Clinical Research Scholar at Moorfields Hospital and as British Medical Association Science Research Scholar, he had been doing clinical investigation with the slit-lamp for some time, yet in examining at the Royal Westminster Eye Hospital only last week five patients referred to him by the Staff, he was able to derive some ten or twelve facts, or comments on facts worth noting for future slit-lamp reports.

After the study of unselected patients, a second line of investigation with the slit-lamp consisted in the study of special and selected cases known to be suffering from some regional affection, and for those who had access to a large clinic, this method was very profitable of results.

A third line of investigation—one which would yield comparatively few results—consisted in the study over a long period of one individual patient known to be suffering from some given alterable condition, and the recording of the features seen with such accuracy that it was possible precisely to define, at each succeeding examination, the changes, often minute, which might have occurred since the previous ex-

amination of that patient. This last method called for laboriously accurate, oft-repeated records, and they required much time, hence it was not a method to be advocated for those who had a large number of cases to select from. In the large clinics of London and the provincial towns there must be a large and wonderful field for slit-lamp research, and there seemed to be scope for an organized combined research in the eye clinics here. And for this, he thought recourse might be had to the team system of investigation, in which the majority of the members of any one team might be post-graduate students who were anxious to gain experience in the use of the apparatus.

An important subject was that of illustrating for publication; every day he felt the material was there if only he had the ability to record it pictorially. He had been much interested in Mr. Rayner Batten's remarks, published in the *Transactions of the Ophthalmological Society of the United Kingdom*, 1923, on the training of draughtsmen for fundus-drawing. Many of those remarks would apply also to slit-lamp pictures, but Mr. Graves thought the training of artists for the latter might take much longer. He had himself tried training two persons, one an artist and one a professional draughtsman, and he had abandoned the attempt, because he had come to the conclusion that it would have taken him a year or possibly two to train either of those persons to see the technical features in quite the same perspective that he saw them himself. He had, instead, decided to paint the pictures as best he could, in the hope of getting pictures which, if not artistic, might at least be technically accurate, and he hoped to supplement his capacity by having elementary lessons in painting. For publishing, he would endeavor to get an artist who would reproduce suitable pictures from his less artistic efforts.

The answer to the criticism that the use of the binocular microscope and slit-lamp took much learning was, that it depended on the aim of its possessor, and that the detecting of many of the most important clinical features early diagnostic of disease soon became self-evident to the possessor and user of his own apparatus. Teaching of the subject took much time, and was somewhat arduous. If it were to be done on a large scale, it would need to be organized.

Mr. Graves concluded by urging certain precautions in regard to this work. There were occasional indications for some degree of restraint or conservatism with regard to the attitude taken towards certain issues of the subject.

Firstly, though no limit was set by convention to the degree of magnification necessary before the use of the term "microscopy" was justified he did not feel sure that the choice of this term for the work had been altogether a fortunate one, because it, and perhaps a natural enthusiasm for a new subject, might account for the somewhat liberal assertions occasionally made as to the possibilities of this work. The subject of the optical section was an anatomical, not a histological one; the latter term was only applicable to the corneal endothelium by the method of specular reflection. He said that not long ago he read an article in which the writer seemed prone to refer to every white speck seen floating in a pathological aqueous as an individual cell, if not as a leucocyte, and to every pigment speck as a pigmented epithelial cell. In one or two cases in which there was indication to open the anterior chamber, Mr. Graves said he had drawn off and examined the aqueous containing these granules, and he would suggest that one should not be tempted, on insufficient evidence, and without careful consideration, too readily to pin histological nomenclature to these new manifestations.

His second precaution was to urge that enthusiasts for the work, while allotting to it its due share of importance, should be careful that it preserved its proper place and its proportional share in the general scheme of the routine clinical examination. It was not good practice to subject a patient to slit-lamp examination to the exclusion of other and preceding methods of examination. In one article on the slit-lamp he read a statement to the following effect: "Now that we have the slit-lamp we are frequently able to explain, in a manner that we could not do before, why a given patient in whom, by means of ordinary appliances, we see no reason why we should not be able to read as far as $\frac{6}{6}$ or $\frac{6}{4}$ can read only to $\frac{6}{9}$ or $\frac{6}{12}$." Mr. Graves suggested that if the ophthalmologist was clinically well trained and was competent with the ordinary appliances, he was able, by using such instruments as a small electric ophthalmoscope, to detect the present of minute imperfec-

tions in the media which were causing obstruction to vision; say, in the cornea, lens, or vitreous. No lens should be studied by means of the slit-lamp unless it had first been scrutinized by close, direct-method ophthalmoscopic examination. If the observer depended only on the slit-lamp examination not only might he fail, though expert with the slit-lamp, to detect some important changes, but his conception of the significance of these changes, from the important point of view of their immediate effect on vision, might be erroneous.

In summarizing the value of the slit-lamp in routine work, Mr. Graves said that in conjunction with the binocular microscope it afforded means, with an ease and precision hitherto unapproached, of detecting and locating and explaining clinical pathological changes some of which were detectable by no other means, many of which were of the greatest diagnostic importance, and all of which were interesting.

He then, by means of the epidiascope, showed illustrations including distortion of the beam owing to alteration of contour near the limbus, in the case of a man who was suffering from paresis of the external rectus following a blow from a stick; nerve fiber conditions; a variety of Hudson's brown-line of the cornea, which had been described by Mr. A. C. Hudson in 1911 under the title "Certain Peculiar Pigmentary Markings in the Cornea," though this feature was usually referred to as Stahli's. He also showed fine changes in the endothelium of the cornea in a diabetic patient; pathological changes in the aqueous; newly formed vessels in the organized exudate about the pupil, due to iritis; changes early in tubercular iritis; water-fissure in the lens of a diabetic patient; changes in senility; injury and dislocation of lenses, and illustrations of vitreous conditions.

BOOK REVIEWS.

I.—**Parenchymatous Keratitis: Interstitial Keratitis: Uveitis Anterior.** By W. T. HOLMES SPICER, London. *British Journal of Ophthalmology*, Monograph Supplement 1.

This essay has the distinctions of having won for the author the Gifford Edmonds Prize in Ophthalmology[1] and of being published as the first monograph supplement of the *British Journal of Ophthalmology*. The author makes no definite statement as to the purpose or scope of the work, but his statement ". . . it behooves us, therefore, to give as complete an account of it" (interstitial keratitis) "as possible while we have it commonly with us," would seem to indicate that he intended the work to be a comprehensive one. Later, however, he refers to it as "these notes." There is no systematic review of the literature, no bibliography is appended, and exact references are seldom given to the few authors mentioned. The reader is left to ascertain for himself whether or not various observations and theories brought forward are new. Dogmatic statements unsupported by evidence are frequently made. These may be true—the reviewer believes many of them are essentially so,—but, on the other hand, none of them may be. The author suggests syphilitic keratitis as a suitable name for interstitial keratitis, but does not himself adopt it.

The author's observations and statistics are based upon unusually extensive material. He says: "These notes are based on an examination of the records of nearly 700 cases. Most of them came under my own observation. Although the records are often incomplete, they nearly all possess features of some value; in the later cases especially, the notes

[1] According to the title page, the essay itself is the prize!

are all my own and are supplemented by sketches indicating the progressive changes as they occurred. In this way I have in my mind progressive pictures of the different kinds of interstitial keratitis, better than could be conveyed by any detailed verbal description."

Presumably the incompleteness of the records is reflected to some extent in the author's statistics. Ninety per cent. of the cases were due to congenital syphilis, 3.3% to acquired syphilis, 6% to other causes. Of the congenital cases 38%, and of the acquired 31% were in males. Of the cases due to other causes 58% were in males. It seems unfortunate that in the other statistics the congenital cases were not dealt with separately. Thus, of the cases with ages over 25, comprising 13.4 per cent., is impossible to determine how many were not due to congenital syphilis. Probably most of them were due to other causes.

There were three cases of infantile syphilis in which interstitial keratitis was present at birth. The question of whether such cases are not really analogous to the interstitial keratitis of acquired syphilis is not discussed.

Following are the statistical tables presented by the author:

AGE OF ONSET.

Under 2 years	4 cases or	0.7 per cent.
From 2–3 years	5 " "	0.9 "
" 3–4 "	7 " "	1.2 "
" 4–5 "	18 " "	3.2 "
" 5–10 "	129 " "	23.2 "
" 10–15 "	118 " "	21.2 "
" 15–20 "	119 " "	21.3 "
" 20–25 "	80 " "	14.2 "
" 25–30 "	48 " "	8.6 "
" 30–35 "	13 " "	2.3 "
" 35–40 "	11 " "	1.9 "
" 40–45 "	2 " "	0.3 "
" 45–50 "	2 " "	0.3 "

In the case of one patient aged 40 "although the choroid had been attacked early in life, yet a primary interstitial inflammation occurred in the cornea at 40. The organism probably had been lying latent in the cornea for 40 years."

MODES OF ONSET OF THE ATTACK.

Peripheral consisting of maculæ at periphery,	155	199 cases
Peripheral consisting of general haze at periphery,	44	58 per cent.
Central disk made up of maculæ	35 cases	10 per cent.
General uniform haze	100 "	29 "
Endothelial cases	12 "	3 "

PRESENCE OF BLOOD VESSELS IN THE CORNEA.

Present without note of position	150 cases	44 per cent.
Present all around the cornea	45 "	13 "
Above and below	135 "	39 "
Horizontally	8 "	2 "

PRECIPITATES ON THE CORNEA.

Punctate form	224 cases	76 per cent.
Plastic deposits including pericentral	70 "	23 "

EVIDENCE OF CONGENITAL SYPHILIS.

Other signs present or family history obtained	70 per cent.
No family history and no other signs found	5 "
No mention of family history or other signs	25 "

Teeth typical or suggestive	44 per cent.
Facial aspect, bony or soft parts	20 "
Bone disease, periosteal nodes, Clutton's joints	5 "
Total deafness	4 "
Deformity of palate	2 "
Wassermann reaction	Insufficient data
Meningitis and its results	1 per cent.
Complete collapse of nose	1 "
Scarring of throat	1 "
Family history	17 "

The average duration until subsidence of congestion was four months. The attacks which ran the quickest course were those among children. Some of the more severe ones lasted only three weeks in each eye and cleared completely. In an adult aged 20, the attack lasted only three weeks and vision was normal in four weeks.

"There are quiet cases in which corneal signs appear and run their course unattended by pain and congestion or dread of light. . . . They are sometimes followed by relapses."

Clearing of the cornea continues for a long period of time.

In one case at the end of the 3rd year V = $\frac{6}{36}$, at the end of the 14th year V = $\frac{6}{9}$. The center of the cornea is the last to clear. Scar lines on the cornea are to a great extent permanent. "Another form of scarring which occurs but rarely consists in an irregular network of fine lines spreading all over the central part of the cornea at a distance below the surface; they somewhat resemble extinct vessels but are densely white; they have a different way of branching, and have little nodes at the point of branching."

"Calcareous films are formed near the surface; they are due to a deposit of salts in the part exposed to evaporation in eyes in which the lymph circulation is already slow." No doubt the well known band keratitis in which lime salts are deposited chiefly in Bowman's membrane, is here referred to. The author points out that these may sometimes be scraped off to relieve irritation and improve vision.

The worst cases are those in which there is vascular invasion combined with large plastic deposits.

"In 75 per cent., both eyes were attacked together or overlapped with no greater interval than 12 months; there were many cases with a long interval: 11, 12 years, and two with 18 years interval; in one of these there was no evidence and no sign of the stigmata of congenital syphilis, the age of the patient at the second attack was 42; in the other case, in which the patient's age at the second attack was 32, there were periosteal nodes. In the case with 12 years interval there were typical teeth, and during the attack in the second eye there was an effusion into the knee joint; the patient's age was 18."

"There is no relationship between the interval and the severity" . . . "There was a greater amount of over-lapping in the young than later in life." "It is not possible to forecast the nature of the attack in the second eye by that of the first." "There were several one eyed cases."

Relapses and recurrences occurred in 9 per cent. of the cases; some of them were quite transient. Several were very severe. In one case the recurrence was 30 years after the first attack. In one case of recurrence after 3 years, there were three definite centers of activity separated by a considerable amount of healthy cornea. "The probable cause of these

local attacks is that the cornea has been incompletely scavenged in the great attack."

In one case a recurrence of an old attack occurred at the age of over 50 years.

"Cyclitis is not always present; in 6 per cent. of the cases careful and repeated examination throughout their course failed to show any signs of it." Choroidal changes are the result of inflammation in the early years of life, but one case is noted of a recurrence of exudative choroiditis eight years after an attack of interstitial keratitis. The characteristic "pepper and salt" fundus is not described.

The author discusses at some length the question as to whether or not an attack of interstitial keratitis can be precipitated by an injury to the cornea, but comes to no definite conclusion. However, he seems inclined to accept this as possible.

He noted five cases with complete history and signs of congenital syphilis which gave them or their offspring no immunity from the acquired disease.

The author observed three cases of interstitial keratitis with locomotor ataxy. Scleritis may occur during an attack or as a form of recurrence; in one case the interval was 8 years. Hypopyon was observed five times, hemorrhage into the anterior chamber three times, and hemorrhage into the cornea twice. "High myopia was noted in 20 cases,—probably a small proportion of the actual cases." The myopia is attributed to weakness of the sclera resulting from antecedent choroiditis. Myopia of moderate degree resulting from changes in the curvature of the cornea following the attack of keratitis, is not referred to.

Only five cases were noted in which the vertical diameter of the cornea was greater than the horizontal. Intraocular tension was low in five cases, secondary glaucoma occurred in five cases.

Probably the most valuable features of the monograph are the author's descriptions and illustrations of the various appearances of the cornea in the hereditary disease. These are evidently based upon very careful and extensive observations. His description of the appearances produced by the involvement of the posterior surface of the cornea is especially noteworthy. It is unfortunate that he was unacquainted with

Igersheimer's experimental work as it would have been of assistance to him in interpreting these appearances.

The fanciful term "Endothelial Catarrh" which he applies to "a wave of opacity advancing over the posterior surface of the cornea," is not likely to meet with general acceptance. In discussing "keratic" precipitates he says "The precipitable matter comes from inflammation of some part of the uveal tract, ciliary body, iris, or choroid; dead cells with coagulable material cast off from the surface pass into the aqueous which becomes loaded with precipitable matter ready to deposit itself on a suitable surface." As a matter of fact, precipitates are not composed of dead cells but of living inflammatory cells. As Fuchs has shown, they consist of clumps of mononuclear cells. Pus cells may collect on the posterior surface of the cornea, but never in the form of precipitates. The author believes it probable "that a certain adhesiveness of the endothelial surface, not necessarily its destruction, due to nutritional changes, must precede the deposit of precipitates." The reviewer has good reasons for believing that precipitates may adhere to normal endothelium, although they are probably more apt to adhere to injured endothelium. "When precipitation is very active the buff-colored deposits in the angle may reach a considerable size, and may become permeated with blood vessels; they may also disappear leaving very faint traces in the angle."

The description of the vascular invasion of the cornea is of great interest. The formation is described of a trunk vessel in the cornea separated at each end from the general circulation by capillaries. "The process is identical with the re-establishment of collateral circulation in a limb when the main artery has been ligatured."

The author's discussion of the pathological histology of the disease is inadequate, as he has overlooked some of the most important cases in the literature. In the one case of the author in which a microscopic examination was made "The cornea had been almost completely destroyed and revealed little of real value." In this case the center of the cornea had broken down and perforated—an extremely unusual occurrence. For this reason it would seem that the eye was worthy of a more complete examination than was evidently given it.

The author's idea of the origin of interstitial keratitis is brought out in the following quotations: "According to our present knowledge the spirochæta pallida is the cause of the attack, and all the manifestations of its growth are due to its presence in the cornea and adjacent parts. It enters the cornea in all probability during the period of its general diffusion throughout the body in the fœtal state and remains there until conditions are favorable for its development. What these conditions are we do not know, but they are generally so timed as to make the attacks fairly simultaneous in the two eyes. Active growth mostly occurs spontaneously; it may be apparently hastened by outside influences, such as deviations from health, injury or other general lowering conditions, but it is only hastened by these influences, it would occur in any case; its coincidence with injury only shows that the time is ripe, for injuries to eyes occur frequently without inducing interstitial keratitis in people in whom the disease subsequently after a long time develops of itself."

"The conclusion seems inevitable; the interstitial keratitis depends on the presence in the eye of the organism of syphilis, the spirochæta pallida, and on its readiness for evolution; if the organism be present interstitial keratitis is practically certain to occur at some time. If the organism be present, but the required state of evolution be not reached, there will be no attack; if the required state be not reached in the two eyes simultaneously there will be an interval between the two eyes; if the state of evolution have been reached there will be an attack in the absence of any stimulus, and in spite of anything that can be done."

"The normal result of an attack of interstitial keratitis is that every part of the cornea in which spirochætes are present and active, is invaded by phagocytes which come through the lymph spaces or are brought by means of blood vessels to the required spots. The object of this phagocytic invasion is to kill the spirochætes: where recurrences take place the scavenging has been imperfectly done, spores of organisms have been left behind, retaining their potential activity, and eventually reaching maturity and developing a fresh attack."

The author does not discuss or even mention the anaphylactic theory, or take into consideration factors of immunity.

According to his view, apparently, organisms in the cornea are gradually undergoing evolution for years, and then within a few days or weeks, reach the proper stage and become violently active. The fact that spirochætes have not been found in affected corneas makes it a highly debatable question as to whether or not they are actually present. The most convincing evidence that they are present is the experimental work of Igersheimer, with which the author is evidently unacquainted, but this cannot be regarded as absolutely conclusive.

"The principles of treatment are to help the development of the disease to its natural ending—the destruction of the spirochætes—it is impossible to cut it short. In severe cases the patient should be kept in bed during the worst part of the attack." "It is above all necessary to maintain the general health and spirits by assuring the sufferers that they will eventually regain the use of their eyes."

"The results of specific treatment were disappointing; it was not uncommon to find cases in which constitutional antisyphilitic treatment had been used during an attack on one eye, and in which the second eye had been attacked immediately after, and had run a more severe course than in the other eye. . . . The records of 45 cases without mercurial treatment show that they did not compare unfavorably with others in which Hyd. c. Cret. or mercurial inunctions had been continuously used." In spite of this, he says "but there is no doubt about its value in some cases." In regard to salvarsan and other arsenical compounds he says "I have found that in the great mass of cases they have no effect, but now and then a remarkable improvement follows their use, chiefly in the reduction of irritability. I have ceased to use this method of treatment in the ordinary run of cases, but still keep it for severe cases where there is much irritability of the eye, and in which the general condition of the patient calls for it." It is difficult for the reviewer to believe that if specific treatment is useless in the vast majority of cases, it is of value in certain cases of the same nature.

It is pointed out that atropine should not be used stronger or more often than is necessary to obtain full dilatation of the pupil. Atropine irritation occurred in 5% of the cases. The author found peritomy, subconjunctival injections of saline

or mercurials, adrenalin, ultraviolet rays, the constant current, and ionization with various drugs, all of no benefit. "Iridectomy I have done in two cases with undilatable pupils and posterior synechiæ; in one case no benefit resulted, in the other some harm." He does not discuss the operative treatment in cases in which there are completely blocked pupils.

The author finds that eyes which have suffered from interstitial keratitis in early life, and later develop senile cataract, bear operation very well.

The author's description of the interstitial keratitis of acquired syphilis is brief and incomplete, and he does not discuss its differential diagnosis with reference to the hereditary disease. "I have not found the acquired disease in the cornea more curable than the hereditary, especially in cases in which a long time has passed since the primary infection." He calls attention to four cases in the literature in which it followed chancre of the lid and says that the coincidence of these two rarities must be in the nature of cause and effect. The reviewer would suggest another explanation, namely, that such cases would be especially likely to be reported. The best way to solve the question would be to ascertain in just what proportion of cases of chancre of the lid interstitial keratitis occurs.

Interstitial keratitis due to tuberculosis is inadequately discussed but the reviewer infers that the author believes, as he does, that tuberculosis never produces a typical picture of syphilitic keratitis. The author recognizes the fact that phlyctenular keratitis and interstitial keratitis may be combined. He gives no statistics relating to tuberculous keratitis.

The discussion of keratitis profunda adds nothing to our knowledge of this condition, and in regard to it also, no statistics are given.

F. H. VERHOEFF.

NOTICE.

Oxford Ophthalmological Congress.

Master, Sir Anderson Critchett, Bart., K.C.V.O.
Deputy Master, Philip H. Adams.
Hon. Treasurer, Robert J. Coulter.
Hon. Secretary, Bernard Cridland, Salisbury House, Wolverhampton.
Past Masters, Robert W. Doyne, Sydney Stephenson.

February, 1924.

Preliminary Notice.

The Oxford Ophthalmological Congress will assemble at Keble College on the evening of Wednesday, July 2nd next, and the Meeting will be held on Thursday, July 3rd, Friday, July 4th, and Saturday, July 5th.

On the morning of Thursday, July 3rd, a discussion will be held on "The General Principles of the Treatment of Convergent Concomitant Strabismus" (with exclusion of the description of details of operations), to be opened by Dr. E. Landolt of Paris and Dr. Ernest Thomson of Glasgow.

Members intending to take part in the Discussion are requested to kindly send in their names to the Hon. Secretary at their early convenience.

The Doyne Memorial Lecture will be delivered on the morning of Friday, July 4th, by T. Harrison Butler, Esq., M.A., M.D., B. Ch. Oxon, the subject being "Microscopy of the Living Eye, with Especial Reference to the Gullstrand Slit-Lamp."

It is proposed at this Meeting to devote one afternoon entirely to demonstrations in the Scientific and Commercial Museums and it is hoped that members will do their best to

bring forward novelties of any kind, *e.g.* pathological specimens, operations, apparatus, etc.

It will be appreciated if those who desire to contribute either papers or demonstrations will kindly notify the Hon. Secretary at their early convenience.

The Official Dinner of the Congress will take place on the evening of Thursday, July 3rd, in the Hall of Keble College.

A General Meeting will be held during the Congress at a time of which due notice will be given in the final program.

BERNARD CRIDLAND,
Hon. Secretary.

Salisbury House,
Wolverhampton.